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THE PROBLEM OF INTRANASAL MEDICATION*

THEODORE E. WALSH, M.D.

AND

PAUL R. CANNON, M.D.

CHICAGO

In these days of health columns in the newspapers and magazines, the public has become aware of the dangers of the common cold and the importance of its early treatment. Unfortunately, however, the proper treatment is not well known, and the layman is advised, through popular advertising, of "certain cures" for colds by the use of nasal drops and sprays. Furthermore, physicians are often asked for their opinion as to the merits of these preparations. Heretofore physicians have assumed, that even if such preparations did little if any good, they did at least no harm. Recent observations of the effects of various drugs on the nasal mucosa and in the lungs following nasal application, however, have proved this idea to be erroneous. It therefore seems important at this time to review the

*From the Departments of Surgery (Division of Otolaryngology) and Pathology, The University of Chicago.

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problem of nasal medication and to point out some of the effects of commonly used drugs, both on the nasal mucosa and on the lungs. Our discussion will consider, first, the action of the vehicles in which the drugs are dissolved, and second, the effects of the drugs themselves. The latter will be considered with particular reference to their antiseptic, astringent and vasoconstricting actions.

THE EFFECTS ON THE NASAL MUCOSA OF MATERIALS USED AS VEHICLES FOR INTRANASAL MEDICAMENTS

1. *Oils:* Perhaps the most commonly used vehicle for intranasal medication at the present time is mineral oil. Its popularity is due, no doubt, to the belief that it is bland and does no harm to nasal tissues, and because it causes no discomfort even in the presence of acute infections. Stark, however, observed that the mucous glands of the rabbit's nose become hyperactive after intranasal use of plain mineral oil. Fox later found in rabbits treated for periods as long as nine months with intranasal sprays of mineral oil, and various drugs dissolved in mineral oil, that the "surface epithelium is desquamated and many polymorphonuclear leucocytes and plasma cells are present. There are a few polymorphonuclears in the substratum. There is a tendency toward new growth of epithelium." He concluded, therefore, that "liquid petrolatum apparently also exerts a deleterious effect on the nasal mucosa of a rabbit when used for nine months." He found even more marked changes when menthol, eucalyptol and camphor were used similarly, dissolved in mineral oil.

In view of the studies of Hilding, Proetz and others demonstrating the importance of ciliary activity in maintaining efficient streaming of mucus in the nose, the demonstration by Proetz and by Lierle and Moore of the different effects of drugs dissolved in mineral oil and watery solutions on ciliary action is particularly significant. The former found that although liquid petrolatum had no immediate effect on the activity of the ciliary beat, it did cause a slowing down or loss of "mucous streaming." He suggested that oily mixtures "are to be avoided where ciliary streaming still functions, because they interfere not with the ciliary beat but with its effectiveness, by lying on the mucous blanket and by being propelled with great difficulty by it." He also concluded that the oil "was not in actual contact with the cilia at all, but separated from it by mucus." When he used 1 per cent menthol or 1 per cent thymol in mineral oil, there was no effect on ciliary activity with the

former, and marked slowing only after 10 minutes with the latter. Lierle and Moore, on the other hand, found that these two drugs in Locke's solution decreased ciliary activity in 11 of 13 animals and that 1 per cent thymol in the same vehicle stopped ciliary activity in from one to three minutes in eight of thirteen animals and caused marked slowing in the other five. These differences, therefore, can be accounted for only by the vehicle in which the drugs were dissolved. It is apparent from these observations that, insofar as their effects on the nasal passages are concerned, oily solutions affect both the mucosa and the cilia deleteriously and that drugs dissolved in them act less effectively than in saline solutions.

2. *Aqueous Solutions:* If oily solutions affect the nasal tissues and cilia adversely, do aqueous solutions also do so? This question has been studied by both Stark and Proetz. The former irrigated the nasal mucous membranes with 0.9 per cent saline, 2.0 per cent saline and plain tap water and found, that although the effect was harmful in every case, isotonic saline solution caused less harm than did either of the other two. Proetz ascertained the effects of various salt solutions on ciliary beat and found that the cilia of both man and animals remained active for long periods in 0.9 per cent solution of sodium chloride, but lost their activity when more concentrated solutions of sodium chloride were used. When the saline solutions were made hypotonic the outlines of the cilia were gradually lost and the surface became cloudy. These experiments indicate quite definitely, therefore, that isotonic saline is the vehicle of choice for drugs to be used in the nose.

THE EFFECTS ON THE NASAL MUCOSA OF DRUGS USED INTRANASALLY

1. *Antiseptics:* Ever since the bacterial nature of disease became known, various chemical substances have been used in the attempt to stop bacterial growth in tissues. This has been particularly true of respiratory disease, and many kinds of drugs have been used in the nose and throat because of their presumed bactericidal effects. In recent years the silver salts, for example arygrol and neosilvol, have been especially popular, and thymol and merthiolate have also been widely used.

Evaluation of the rationale of such medications requires consideration of several factors: first, are such drugs actually bactericidal; second, do they remain in the nose long enough and in proper

concentration to exert an effect; and third, do they injure nasal mucosa, mucous glands or cilia? In order to ascertain to what extent these drugs are bactericidal for micro-organisms commonly found in respiratory infections, we performed an experiment similar to one described by Archibald and Brown in their study of the bactericidal effects of lipiodol. Pus from a patient with bronchiectasis was mixed thoroughly with solutions of the drugs to be tested and the mixtures were allowed to stand at room temperature. At varying intervals of time, 0.1 cc. of the mixture was mixed with melted blood agar, plates were poured, and after 24 hours incubation, colony counts were made. The results are shown in Table I, where it is apparent that the only drug with any appreciable bactericidal effect was 1 per cent thymol. When one considers that in an upper respiratory infection the causative organisms are not lying free on the surface of the mucous membrane, but are in the epithelial or subepithelial tissues, and furthermore, that in all probability drugs placed in the nose remain there for no longer than from ten to fifteen minutes, it is improbable that even such drugs as thymol can exert much bacterial effect in the upper respiratory tract.

Lierle and Moore, and Proetz have also investigated the effects of the silver salts, of thymol and merthiolate on the ciliary beat. They all agree that thymol causes immediate cessation of the ciliary beat, and that merthiolate causes an initial slowing of ciliary action, with cessation after a few minutes. Lierle and Moore state that the silver proteins cause initial increase of ciliary activity followed by a marked slowing, although they suggest that this latter effect may have been due to the fact that the drugs were used in watery (not isotonic) solution. Proetz noted no effect on the cilia from the application of 10 per cent argyrol, but observed an interference with the streaming of mucus, which he interpreted as being due to the clumping of the precipitated drug on the surface of the membrane.

2. *Astringents:* Astringents are also frequently used in nasal therapy, and their use has become more general in recent months because of the evidence that experimental poliomyelitis of monkeys, produced by intranasal instillation of the virus, can be largely prevented by a previous spraying of the noses of these animals with astringent solutions (1 per cent tannic acid, 2 per cent zinc sulphate, 4 per cent sodium aluminum sulphate). The effect of these drugs is entirely nonspecific, the purpose of their use being to cause the

TABLE I

Antiseptic	1 hr.	16 hrs.	24 hrs.	44 hrs.
Neosilvol 5%	+	+	+	+
Neosilvol 10%	+	+	+	+
Argyrol 5%	+	+	+	+
Argyrol 10%	+	+	+	+
Thymol 1%	10	5	0	0
Menthol 1%	+	+	+	+
*Isedrin Comp.	+	+	+	+
Saline	+	+	+	+

*Isedrin Compound (Lilly) is a watery solution of ephedrine made istonic by the addition of dextrose and containing merthiolate 1:5,000.

+Innumerable colonies.

deposit on the nasal tissues, particularly over the olfactory area, of a protein precipitate which will act as a barrier to the passage of poliomyelitic virus from the nasal passages into and along the olfactory nerves. Because of these experiments, large numbers of children have been treated with nasal sprays of tannic acid and zinc sulphate as a prophylactic measure against poliomyelitis, but the end results of this type of treatment have not been sufficiently investigated as yet to determine its success. Clinically, there have been reports of the loss of smell following the use of tannic acid and zinc sulphate sprays, and in view of the uncertainty at the present time that the nose is actually the portal of entry in poliomyelitis, the use of such astringents seems questionable.

But little information is available as yet as to the local effects of astringent solutions. Lierle and Moore, however, have found that 2 per cent zinc sulphate caused almost immediate cessation of ciliary activity in the rabbit's nose and that resuscitation was not effected.

3. *Vasoconstrictors:* Probably the most common complaint of patients suffering from upper respiratory disease is nasal obstruction,

and nearly all of the solutions prescribed for use in the nose contain a vasoconstrictor. The most widely used drugs for this purpose are adrenalin, ephedrine, benzedrine and neosynephrine. It is important, therefore, to know what effect these drugs may have on the mucous membrane and the ciliary activity. All observers who have worked with adrenalin agree that this drug in a 1:1000 dilution causes immediate cessation of ciliary action. Weak saline solutions of ephedrine, on the other hand, cause an initial speeding of the ciliary beat at times (Lierle and Moore), but this is in no way detrimental to ciliary activity (Proetz and Lierle and Moore). Fitzhugh reports that neosynephrine hydrochloride causes no apparent harm to the nasal mucosa, and states that this drug has a slightly longer vasoconstrictive effect than either adrenalin or ephedrine and is less toxic than either. Benzedrine is recommended for intranasal application either in solution in oil or in the form of a vapor. There is no report on the effect of the drug in watery solution on the nasal mucosa or cilia; it is, however, a powerful central nervous stimulant and should be used only with the greatest caution.

In view of the fact that the maintenance of the physiological functioning of the nasal mucous membrane and the promotion of adequate drainage from the nasal sinuses are the two essentials in the treatment of upper respiratory infections, weak saline solutions of ephedrine and neosynephrine appear to be the medications of choice for intranasal application.

THE PASSAGE OF NASAL MEDICAMENTS FROM THE NOSE TO THE LUNGS

Of more importance than the effect of nasal medicaments upon the tissues of the upper respiratory tract is the problem of their effect upon the lungs. All doubt as to the ease with which medicinal solutions placed in the nose can go directly to the lungs has been removed by the observations within recent years of the widespread occurrence of lipoid pneumonia secondary to the use of nasal oil drops and sprays. This condition was first recognized in a definite way by Laughlen in 1925, who described the changes in the lungs following the aspiration of oils, given either as intranasal medication or orally, and reported five cases, four in children and one in an adult. Since his report many others have appeared in the literature, with increasing frequency in recent years. Ikeda, in 1937, found 106 cases recorded in the literature by twenty-four authors in the ten years from July, 1925, to April, 1936. His conclusions from

his study were as follows: "(1) There is increasing incidence of lipoid pneumonia of both types in adults, 21 of the 38 cases in adults having been reported within the last year of the period under survey, eighteen of the infantile and three of the adult type; (2) liquid petrolatum is practically the sole etiologic agent; (3) only in 14 per cent of the total number of cases, or 39.5 per cent of the cases in adults, was the condition recognized as lipoid pneumonia of the adult type; (4) in these cases males predominated three to one over females; (5) the condition usually occurs in the fifth decade or later; (6) application of oil in the upper air passage as drops, spray, etc., and direct intratracheal instillation are the most prevalent modes of aspiration, and (7) aspiration over a period of years is an important etiologic factor." He described the infantile type as "interstitial proliferative inflammation not infrequently combined with exudative pneumonia" and the adult type as "chronic granulomatous pneumonitis" and believes that the former can be caused by any kind of oil, whereas the latter is due most frequently to mineral oil. Although many of the cases in infants have been due to aspiration of codliver oil or milk and cream, and many in adults have followed the ingestion of mineral oil by debilitated patients or those with some defect in deglutition, nevertheless, of all the cases of lipoid pneumonia thus far reported, approximately a third have been associated with the intranasal use of medicated oils. Holt and McIntosh, in their text-book, Diseases of Infancy and Childhood, reported 23 cases of lipoid pneumonia seen at the Johns Hopkins Hospital in seven years, and remark "the condition is not amenable to treatment. It occurs with sufficient frequency to make one hesitate before prescribing oil applications to the nose and throat."

The intranasal use of medicated oils, therefore, may be regarded as a widespread natural experiment which has proved that such light fluids may go directly to the lungs and cause serious irritation, the severity depending upon the kind and quantity of material aspirated and the length of time during which it has been used. In the past year we have observed three cases of lipoid aspiration in adults at the University of Chicago Clinics, all due to aspiration of oil from the nose and nasopharynx. Of these three patients, it should be noted that two were adults in good health and leading active lives. The following brief protocols from the necropsy reports illustrate the general findings.

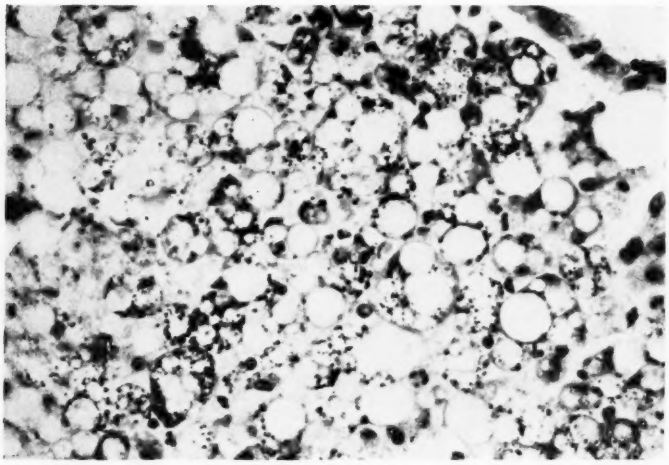


Fig. 1. Photomicrograph from the lung of patient (Case 1) who had used nasal drops of liquid albolene for two years prior to death. Note macrophages filled with oil droplets and dust. x575

CASE 1.—Male, age 71, a physician in active practice, died presumably from an overdose of morphine, self administered. He had been in the habit of using liquid albolene in the nose each night for several years preceding his death.

From the necropsy report by Dr. Dunlap: "The lower lobe of the right lung is consolidated centrally while a few areas on the surface are crepitant. The upper lobe is consolidated in its upper part. Creamy white pus runs from the bronchi on the cut surface of the lung."

Histologic report: "The lung tissue is loaded with macrophages containing large and small fat droplets and granular brown pigment. There are many large fat droplets which are extracellular. The lung tissue shows marked fibrosis and is infiltrated with round cells and many multi-nucleated giant cells containing fat.

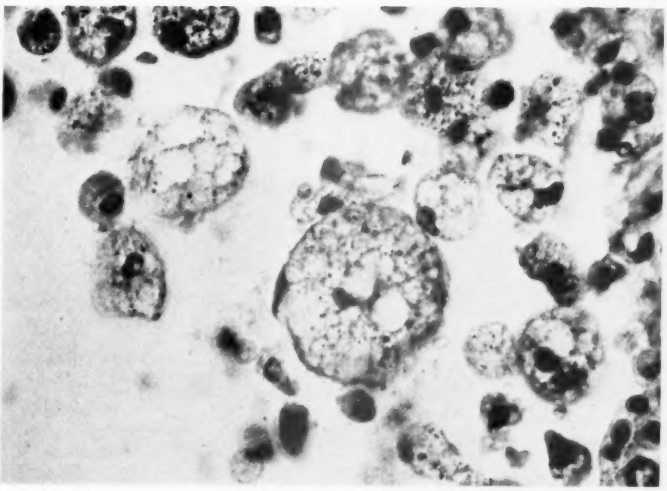


Fig. 2. Photomicrograph of lung of patient (Case 2) who had used "oil drops" intranasally at intervals for many years. Note the large cells filled with oil and dust particles. x1050

CASE 2.—Male, age 40, had a history of chronic mental depression and chronic alcoholism for many years, but had been active in his work until his death. Death was due to a bullet fired by the patient into his head. He had suffered from dryness of the nose for some years and had used menthol and albolene nose drops. He also had been in the habit of frequently swallowing a teaspoonful of white vaseline in order to relieve the dryness of his throat.

From the necropsy report by P. R. Cannon: "Both lungs are heavier than normal, are moderately distended and do not collapse. The posterior half of the left upper lobe and the posterior two-thirds of the left lower lobe are airless and solid and the overlying pleura is covered with fibrin. Only the anterior surface is vesicular. The bronchi on the cut surface exude whitish pus. At the base of the left lower lobe extending across the entire lower margin is a series of abscesses varying in size from a few millimeters to one centimeter in diameter. The right lung shows similar changes but not as marked as the left."

Histologic report: "Sections through the bases of the lower lobes, particularly the left, show multiple acute abscesses, many of which are confluent and contain bacterial masses. There is marked necrotising bronchitis. Surrounding the abscesses, the alveoli are filled with plugs of fibrin and coagulated albumin. In many of the alveoli are large pigmented dust cells also containing large vacuoles which with fat stains are seen to be lipoid."

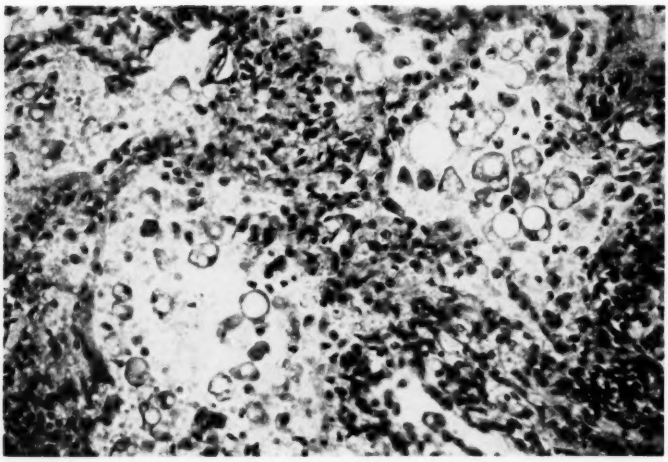


Fig. 3. Photomicrograph of lung of patient (Case 3) who had received mineral oil and cotton seed oil as eye drops. Note the thickening of the alveolar walls and the large macrophages filled with oil droplets lying loose in the alveoli. x320

CASE 3.—Female, age 14, was admitted to the hospital in coma. She had a brain tumor and was in hospital for seventeen days before her death. During this time she received no food by mouth, being fed entirely intravenously and per rectum. During her stay in the hospital she developed acute conjunctivitis and eye drops of cotton seed oil and of mineral oil were recommended. She received one drop of cotton seed oil in each eye every two hours for seven days and the same dose of mineral oil for the subsequent nine days.

Histologic report (P. R. Cannon): "Sections of the lungs show mainly simple edema and patchy early bronchopneumonia with marked engorgement of the interalveolar capillaries. Many of the macrophages contain large vacuoles suggesting lipoid ingestion."

The effects upon the lungs of aspirated oils as seen at necropsy tell little about the early action of such substances; this can only be studied in experimental material. Pinkerton, in 1928, injected various oils intratracheally into rabbits and puppies and examined the lungs at intervals up to five and a half months. He found that when vegetable oils were used there was very little reaction, and that the oil lay free in the alveoli and had all disappeared in five and a half months. Such lungs, both grossly and histologically, appeared normal. With mineral oil the lungs showed "marked consolidation of the involved portions, but the degree of firmness increased progressively with the length of time the oil had remained in the lungs." Microscopically, the alveoli of the involved portions were filled with large numbers of mononuclear phagocytes, each containing many small oil droplets. He pointed out that the difference between vegetable and mineral oil is that the latter is taken up by the mononuclear phagocytes while the former lies in the alveoli entirely extracellular. Ikeda, in 1935, experimenting with rabbits, found that vegetable oils were on the whole less irritating than animal or mineral oil, but that it was "consistently found that such vegetable oils as were employed, namely, castor oil, olive oil and lipiodol, all induced a slight degree of proliferative reaction and the invasion of macrophages in small numbers about the retained oil drops."

We wished to ascertain the early effects upon the lungs, particularly within the first few days, of medicated oils introduced into the nostrils of normal, "snuffles-free" rabbits. The oil was given intranasally by means of a tuberculin syringe without a needle, in doses in most instances of 1 cc., 0.5 cc. per nostril. The rabbits were usually in a sitting position in a rabbit box, and under these circumstances the oil almost invariably gravitated to the anterior pulmonary lobes. If the rabbits were held in a vertical position the lower or posterior lobes were most affected. The animals were killed in two or three days after instillation of the oil, the lungs fixed *in situ* with Zenker-formol solution, after which celloidin sections were prepared and stained with hematoxylin and eosin. The following medicaments were used: plain mineral oil, Vick's Vatronol, mistol, pinoleum, benzedrine in oil, chloretone inhalant and oliodin. The changes observed in the lungs were essentially similar for all preparations and were as follows: Within twenty-four hours there was a focal edema of the involved portions of lung, with extensive desquamation of septal cells, with many filled with large oil vacuoles. The alveolar walls were thickened and in such areas there was definite focal atelectasis. In some instances there was associated acute bronchitis, although usually this was absent. The histologic picture was

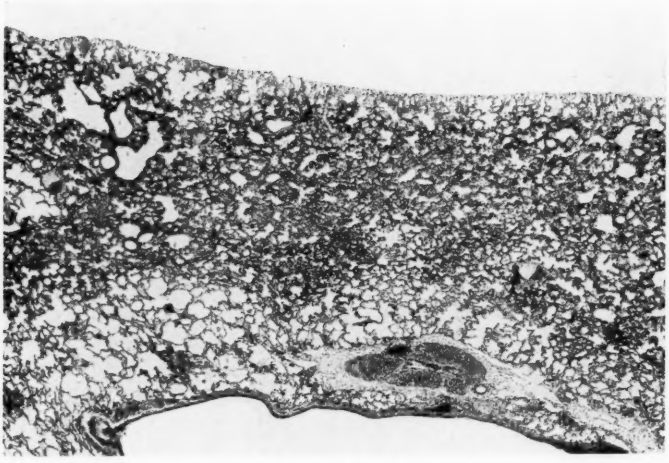


Fig. 4. Photomicrograph of the lung of a rabbit which received 0.5 cc. chloretone inhalant in each nostril once. The animal was sacrificed three days later. Note the extensive area of consolidation. x35

constant and showed definitely that such medicated oils, and even plain mineral oil, are toxic enough to pulmonary tissue to cause increased permeability of capillaries and leakage of albuminous fluid into the air spaces. Figs. 4, 5 and 6 illustrate the characteristic findings.

When the oil was given in large total amounts and over a longer period of time, lesions in the lungs could be demonstrated in roentgenograms. Fig. 7 shows the roentgenogram of a rabbit before and after the intranasal administration of a total of 18 cc. of oliodin in seven days. This experiment was undertaken because the dose of this oil, recommended by the manufacturers for children from six months to ten years of age is "one-half dropperful in each nostril three or four times a day." Each dropperful contained 1.5 cc., making a total dose of 4.5 cc. to 6.0 cc. per day. The total dose that might be given to a six-months-old child in a week would be from 30 cc. to 40 cc. It seemed that 18 cc. in a week for a rabbit, therefore, was not incomparable. The roentgenogram shows an area of consolidation in the right lower lobe with the heart pulled to the right side. Fig. 8 shows a section from the right lower lobe of this animal, where one can see the numerous lipophages filling the alveoli.

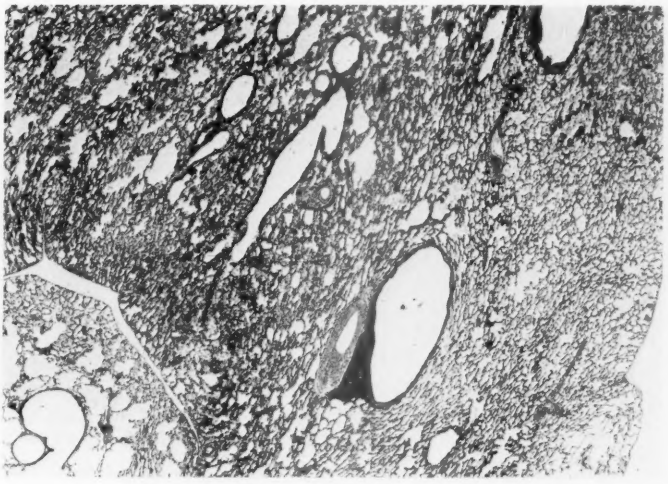


Fig. 5. Photomicrograph of the lung of a rabbit which received 0.5 cc. mintel in each nostril once. The animal was sacrificed two days later. Note the consolidation around the bronchi and the adjacent edema. x35

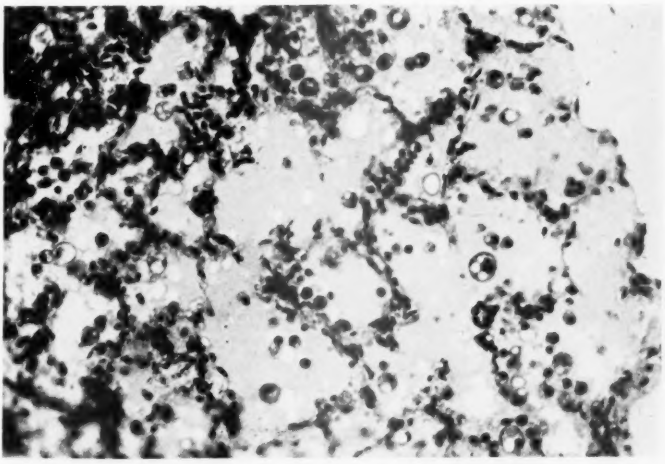
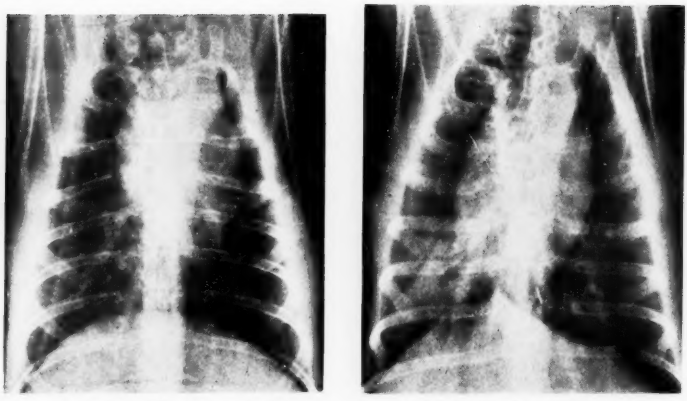


Fig. 6. Photomicrograph of the lung of a rabbit which received 0.5 cc. of benzedrine in oil in each nostril once. Killed in three days. Note the edema, lipophages and desquamation of septal cells. x320



A. B.

Fig. 7. Roentgenogram of the chest of a rabbit that received 1.5 cc. oliodin intranasally twelve times in seven days. *A*, before, *B*, after, the treatment. Note the wedge-shaped consolidation in the right lower lobe and the displacement of the heart to the right.

It should be re-emphasized that these experiments were all performed in normal rabbits free from upper respiratory infection. The nasal oils, when used clinically, however, are employed because of rhinitis or upper respiratory disease, and in passing through the infected areas are liable to carry down living micro-organisms to the lungs. In order to determine the combined effect in the lungs of nasal oils and living bacteria therefore, we instilled medicated oil into a normal rabbit, and 24 hours later instilled a suspension of living micro-organisms cultured from a rabbit with snuffles. Fig. 9 shows the granulomatous lesions distributed throughout both lungs, which were present twenty days after the treatment.

THE EFFECTS UPON THE LUNGS FROM THE INTRANASAL ADMINISTRATION OF WATERY SOLUTIONS OF SOME COMMONLY USED MEDICAMENTS

In view of the ease with which the nasal oils may pass from the nose to the lungs, the question arises, do watery solutions of drugs applied in the nose also reach the lungs, and if so, do they cause harm? The following experiments were undertaken in an attempt to elucidate this question.

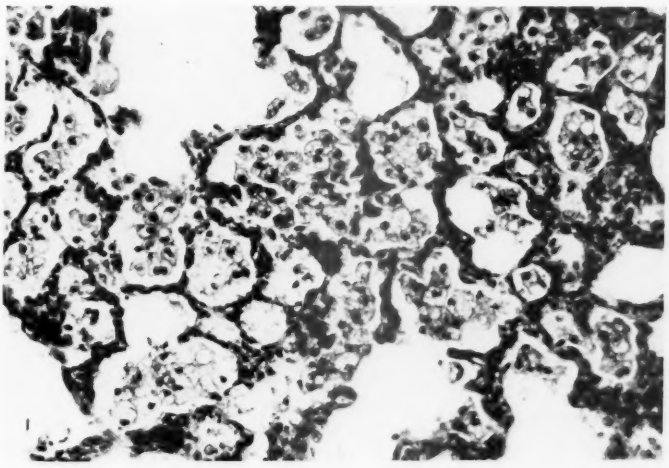


Fig. 8. Photomicrograph of a section from the right lower pulmonary lobe of rabbit which received 1.5 cc. of oliodin twelve times in seven days. Note the marked thickening of the alveolar walls and the filling of the alveolar spaces with oil-filled macrophages. x275

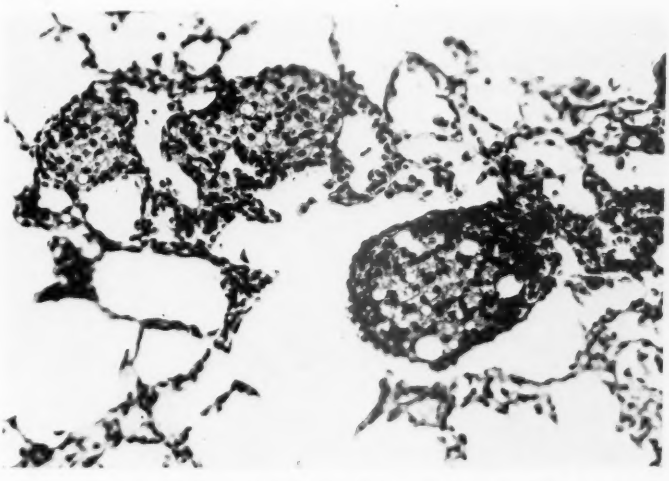


Fig. 9. Photomicrograph of the lung of a rabbit that received 0.5 cc. mistol in each nostril once, followed next day by an equivalent dose of a saline suspension of living organisms. The animal was sacrificed twenty days after the first treatment. Note the granulomatous lesions containing oil droplets. x325

1. *Antiseptics:* It is generally assumed that silver salts, neosilvol and argyrol, which are so widely used in nasal therapy, are non-toxic and non-irritating. If this be true, their intranasal use, even if followed by passage into the lungs, should cause no damage to pulmonary tissues. The following observations, however, suggest that they may be potentially dangerous when so employed.

Healthy rabbits were treated with these drugs in the same way as those which received the nasal oils, and were studied histologically and by roentgenogram. Figs. 10, 11 and 12 are photomicrographs from them; the extensive edema, necrosis and desquamation of septal cells, all demonstrate the toxic effect of such materials when they enter the lungs under the conditions of these experiments.

When such animals received 0.5 cc. per nostril at weekly intervals over a period of several weeks, bronchopneumonic lesions could be demonstrated by roentgenograms. Fig. 13 is the roentgenogram of a rabbit which received 0.5 cc. of 5 per cent neosilvol per nostril at weekly intervals for sixteen weeks. Two weeks later this animal was injected intravenously with 5 cc. of a 5 per cent suspension of colloidal mercuric sulphide, was killed by air embolism 2 hours later, and the lungs cleared in oil of wintergreen by the Spalteholtz method, in order to demonstrate the accumulation of particulate materials in the areas of pulmonary inflammation. Fig. 14 shows the photograph of these lungs after this preparation. Similar lesions were seen in all of 19 animals treated with 5 per cent argyrol or 5 per cent neosilvol over the same period of time.

Since such marked changes occurred in the lungs of normal, healthy rabbits after the intranasal use of neosilvol and argyrol, we wished to determine the effect of such medication in rabbits suffering from "snuffles." The following protocol illustrates what happened in one such case.

Protocol 1. An adult rabbit suffering with snuffles, with a purulent discharge from the nostrils, received 0.5 cc. of 10 per cent neosilvol in each nostril on October 25, 26 and 29, 1937. Following these treatments the animal's condition remained unchanged so far as the nasal discharge was concerned, he ate well and gained weight. On February 9 and 10, 1938, he received 1.0 cc. of 10 per cent neosilvol in each nostril, following which the nasal discharge increased markedly; he refused food, developed a cough and lost weight. He was killed one week after his last treatment. Necropsy revealed a markd pleural thickening over both apices with consolidation throughout the lungs. Fig. 15 shows the gross specimen of the lungs and Fig. 16, the photomicrograph of an area of consolidation. The pneumonic consolidation and the purulent bronchitis are apparent.

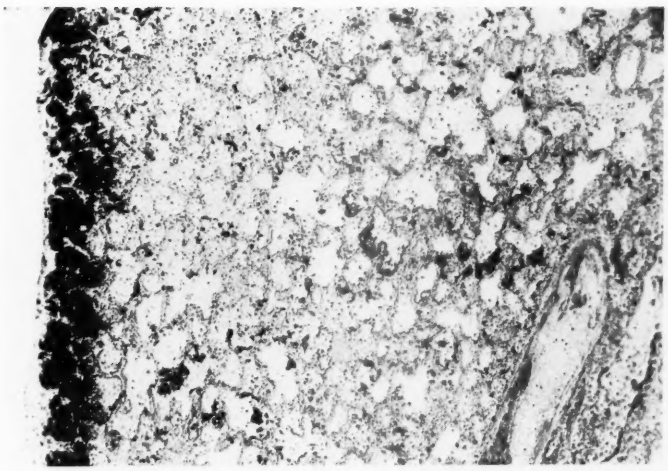


Fig. 10. Photomicrograph of the lung of a rabbit treated with 0.5 cc. argyrol, 10 per cent, in each nostril once. The animal was sacrificed on the third day after the treatment. Note the edema and desquamation of the alveolar cells and the silver deposits beneath the pleura. x135

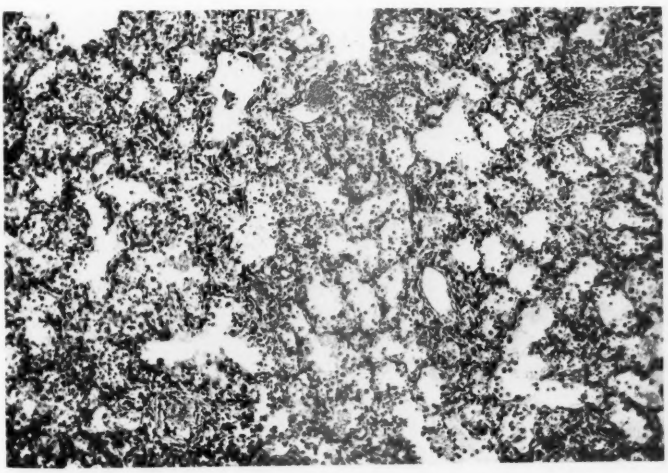


Fig. 11. Photomicrograph of a section of lung from a rabbit that received 0.5 cc. of 5 per cent argyrol in each nostril once. Killed at 48 hours. Note the pulmonary edema and the desquamation of septal cells. x120

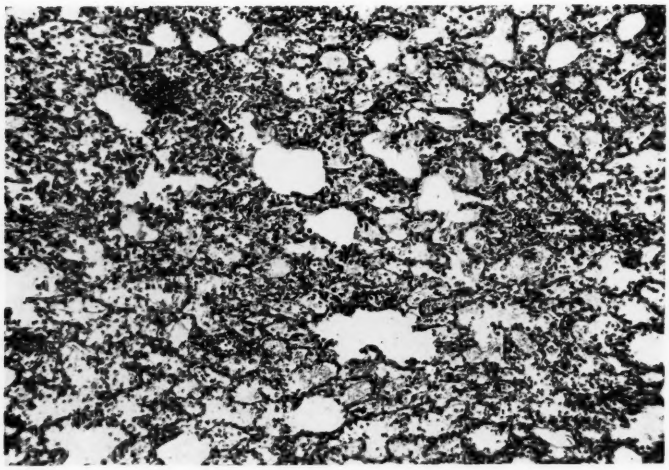


Fig. 12. Photomicrograph of a section of lung from a rabbit which received 0.5 cc. of neoefemist in each nostril once. Killed 48 hours after treatment. Neoefemist contains 5 per cent neosilvol. Note the edema, thickening of the alveolar walls and the desquamation of septal cells. x125

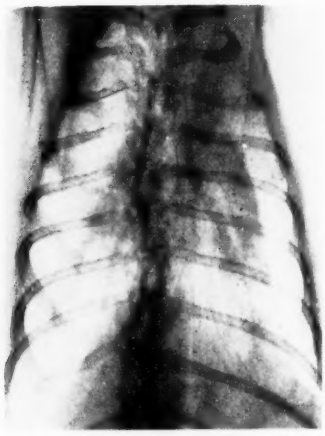


Fig. 13. Roentgenogram of the lung of a rabbit that had received 0.5 cc. of 5 per cent neosilvol in each nostril once a week for 16 weeks. Note the consolidation in the left upper and right middle lobes.

Two healthy "snuffles-free" rabbits were also treated as follows: One received in each nostril 0.5 cc. of a saline suspension of living micro-organisms, cultured from a rabbit with snuffles, on December 21, 22 and 23, 1937. The other animal received equal parts of the same suspension and 5 per cent neosilvol, 0.5 cc. per nostril on the same days. Roentgenograms were taken before treatment and again on January 5, 1938, fourteen days after the last treatment. The animals were killed on this day. Fig. 17 shows the roentgenogram of both animals, *A*, before, and *B*, after the treatments. No lesion is demonstrable in the animal receiving bacterial suspension alone, whereas a definite area of consolidation can be seen in the left lower lobe of the animal receiving the mixture of neosilvol and bacterial suspension. Fig. 18 shows the photomicrograph from the rabbit which received only the bacterial suspension, and Fig. 19, that of the animal receiving the mixture. The marked differences in the lesions are apparent, with only a proliferative pneumonia in the former and with suppurative bronchitis and peribronchial necrosis in the latter.

2. *Astringents:* As stated above, several astringents have been used as sprays in an attempt to prevent the development of poliomyelitis. Although we know of no evidence that such solutions ordinarily get into the lungs, the possibility exists that they may under some circumstances. For that reason we wished to determine the effects of some of these astringent solutions upon the lungs of normal rabbits. The following solutions were instilled intranasally, 0.5 cc. per nostril: 1 per cent tannic acid, 2 per cent zinc sulphate and 4 per cent sodium aluminum sulphate. The animals were killed in from one to three days. Figs. 20 and 21 show the effects under these conditions, viz., pulmonary edema, necrosis and desquamation of macrophages. We conclude, therefore, that such materials are potentially dangerous, if or when they get into pulmonary tissues in sufficient quantity or concentration.

THE EFFECTS UPON THE LUNGS OF VASOCONSTRICTIVE AGENTS IN WATERY SOLUTION

In the course of our studies we have tested aqueous solutions of some of the commonly used vasoconstrictive agents, instilled intranasally into normal rabbits. In no instance have we found any evidence of severe pulmonary irritation or necrosis such as we have seen so constantly after the use of nasal oils, antiseptic solutions or astringents. Ephedrine, 0.5 per cent in normal saline, neosynephrine, 0.25 per cent in normal saline, and isedrin compound have been used; lungs from such animals contained no demonstrable lesions. Such drugs, therefore, apparently act upon vasomotor mechanisms but at least not to a significant degree upon pulmonary tissues as toxic agents.

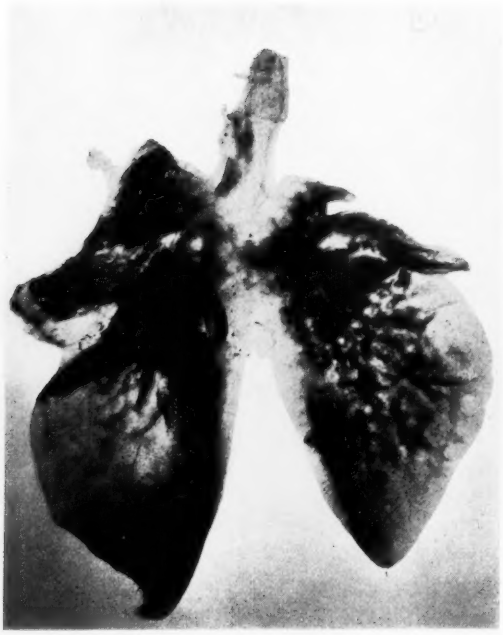


Fig. 14. Photograph of the lungs of a rabbit that had received 1 cc. of 5 per cent neosilvol intranasally once a week for sixteen weeks. Two weeks after the last dose the animal received an intravenous injection of mercuric sulfide and was sacrificed two hours later. The lungs were cleared in oil of wintergreen by the Spalteholtz technique. The mercuric sulfide accumulated in the inflammatory areas.

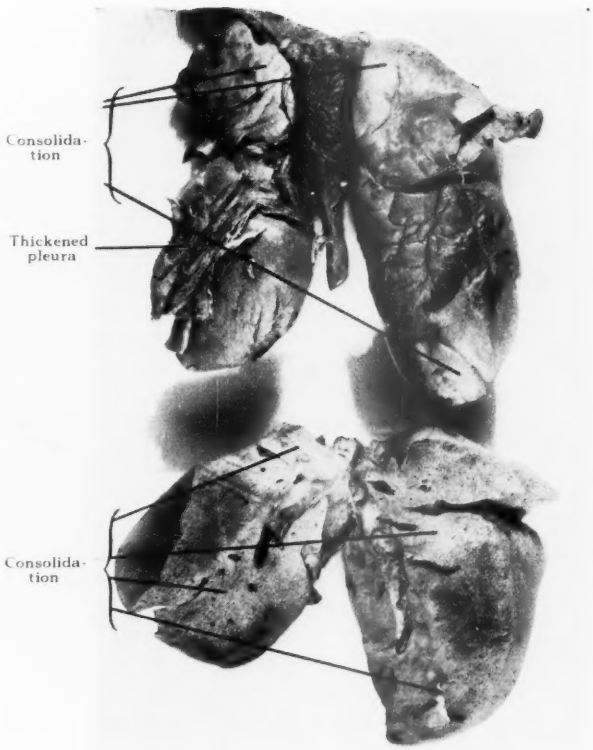


Fig. 15. Photograph of the lungs of a rabbit suffering from snuffles, after treatment with neosilvol 10 per cent (protocol 1).

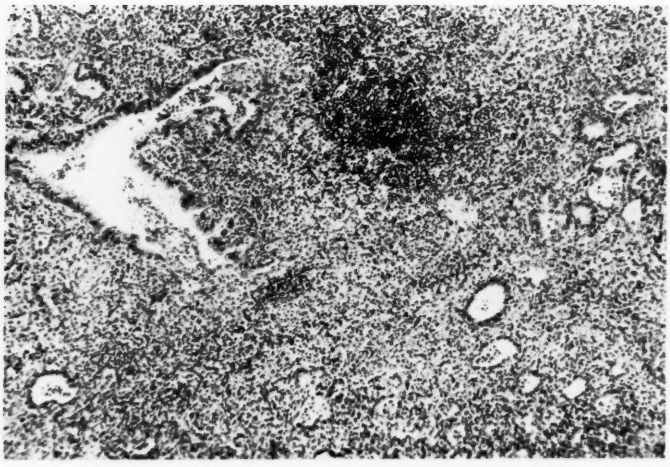
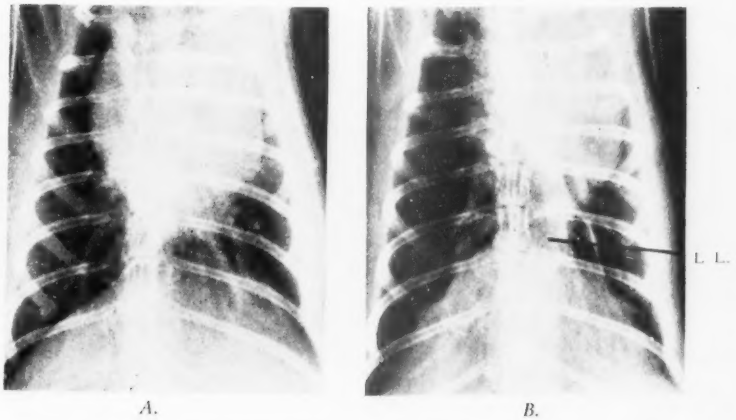


Fig. 16. Photomicrograph of the lung from rabbit described in protocol 1. Note the suppurative bronchitis and the extensive atelectasis and pneumonic consolidation. x125

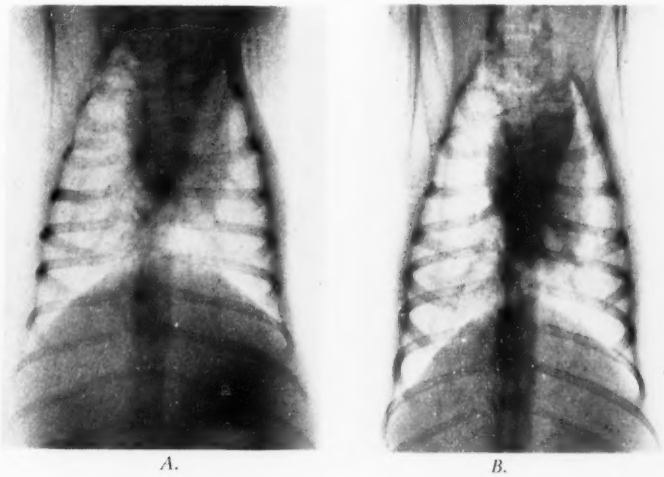
COMMENT

These studies emphasize particularly the potential dangers from the use of medicated oils and also antiseptic and astringent solutions, because of the chance that such materials may go directly from the upper respiratory passages to the lungs and there cause serious damage to pulmonary tissues. Objection may be made that even though they do so in lower animals, as for example in the rabbit, they do not necessarily do so in human beings. This argument, however, fails with respect to the nasal oils, inasmuch as the increasing numbers of cases of lipoid pneumonia now appearing in the literature proves that such light fluids readily enter the lungs in both infants and adults. It is reasonable to assume, therefore, that aqueous solutions of antiseptics, when similarly used in the nose, may also reach the lungs, particularly since we have shown that in the normal rabbit, they get to the lungs as easily and as directly as do the nasal oils. It would seem more important to prove that they do not enter the lungs when used in humans, than to assume that they do not. The possibility exists that if these aqueous solutions of antiseptics get into normal pulmonary tissues and cause pulmonary edema and necrosis, this devitalizing action may set the stage for the development of pneumonia, if and when potentially pathogenic bacteria enter such areas.



A. B.

Fig. 17. Roentgenograms of a rabbit that had 0.5 cc. of equal parts of neosilvol and suspension of living organisms in each nostril three times. Note the increased density in the left lower lobe. (L. L.)



A. B.

Fig. 17. Roentgenograms of a rabbit that received living bacterial suspension only.

It would seem that too little attention has been given to the question of the rationale of intranasal therapy with medicated solutions. Is there, for example, any substantial evidence that either the medicated oils or the aqueous solutions of antiseptic agents actually exert a significant bacteriostatic or bactericidal effect, and if they do, is there any evidence that they can get into or beneath the epithelium where the infecting organisms are growing? Is there any evidence, furthermore, that, with the mucous blanket being renewed so frequently, a solution instilled or sprayed once or twice in a day, will remain localized long enough to cause a therapeutic effect? Certainly the experiments of Hilding cast much doubt upon this latter possibility. Our experiment with bronchiectatic pus failed to show any bactericidal effect by the silver protein solutions or by merthiolate. Although 1 per cent thymol was bactericidal, it should be noted that this solution also causes almost instantaneous cessation of ciliary activity, indicating that it must be a powerful protoplasmic poison.

Even though such medicated materials are neither bacteriostatic or bactericidal in the nasal passages, the question still remains as to whether they may cause harm to cilia and mucous membranes or may interfere with normal mucous flow. There are occasions, of course, when the good may outweigh the harm; for example, although the topical application of cocain has been shown to stop ciliary activity, the value of this drug in some intranasal surgical procedures has been amply demonstrated. Studies of the local effect in the nasal passages from the use of oils, antiseptics and astringents are still quite incomplete, but several have been mentioned above which indicate that such materials may injure cilia, may induce inflammation of the nasal tissues and interfere with normal "mucous streaming." Histological studies, however, as to the local effects on normal tissues, from the instillation or spraying of antiseptic solutions and astringents are still lacking.

It is in the lungs that their effects are perhaps best known. But even here there is but little evidence as to the possible dangers from the pulmonary aspiration of watery solutions of antiseptics and astringents. The manufacturers of one of the silver protein salts say in their advertisement that the solution is "non-toxic, definitely bacteriostatic, and above all, it is markedly soothing to inflamed tissues." Our experiments proved, on the contrary, that these solutions were exceedingly toxic to the pulmonary tissues of normal healthy rabbits, that they had no antiseptic effect upon purulent material from the bronchiectatic cavity of a human patient, although 1 per cent thymol did, and that they caused, not a soothing effect, but necrosis

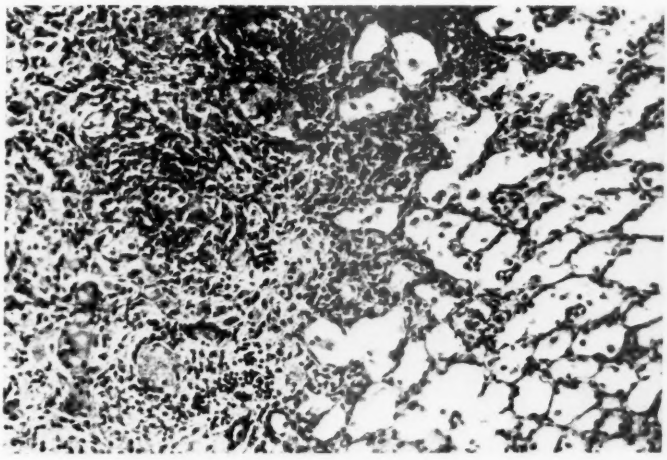


Fig. 18. Photomicrograph of the lung of a rabbit that had received three doses of 1 cc. each on successive days of a saline suspension of living organisms cultured from a rabbit with snuffles. Sacrificed 14 days after the last treatment. Note pneumonic consolidation and desquamative alveolitis but no evidence of necrosis or abscess formation. x300

of normal pulmonary tissues of rabbits. It is doubtful that substances so toxic to the tissues of one species of mammal would have the opposite effect in another. We infer, therefore, that such materials should be used intranasally with great caution, if at all, lest some of them get into the lungs and cause chemical injury. The danger would appear to be all the greater when they are used in the presence of acute upper respiratory infection, because of the possibility that pathogenic micro-organisms might be carried by them into the lungs and into an area of decreased resistance induced by the chemical agent. This hazard would seem to be particularly great in infants and young children lying in bed. It should be remembered that a bronchopneumonia arising under such circumstances would obscure all evidences of the initial injury; indirect proof may be possible by the chemical analysis of such lungs for the quantitative determination of silver, but so far as we know, this has not been done.

Aside from the fact that the intranasal use of astringent solutions has caused loss of smell and interference with ciliary action, but

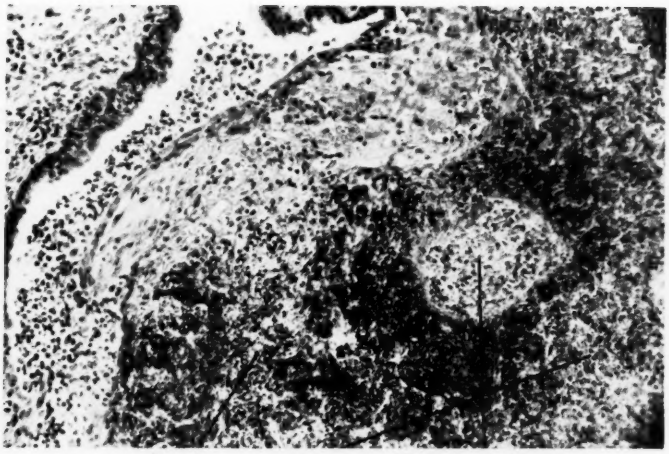


Fig. 19. Photomicrograph from the lung of a rabbit that received three doses of 1 cc. each of a mixture of equal parts of 5 per cent neosilvol and a saline suspension of loving organisms from a case of snuffles. The animal was sacrificed fourteen days after the last treatment. Note the large bronchus filled with pus, the destruction of the bronchial epithelium and the marked peribronchial necrosis. x250

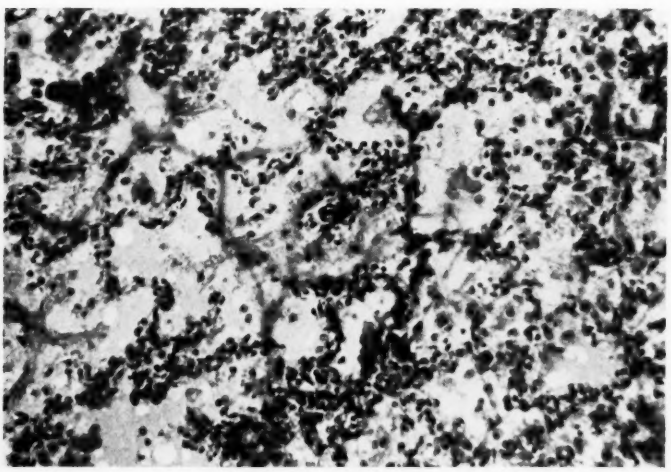


Fig. 20. Photomicrograph of a lung of a rabbit that received 0.5 cc. of 0.5 per cent zinc sulphate in each nostril once. Sacrificed three days after the treatment. Note the marked edema and the desquamation of alveolar cells. x250

little study has been made of their local or pulmonary effect. Nevertheless, the demonstration that these solutions are toxic to mammalian pulmonary tissues suggests that they should be used with caution as intranasal sprays, even though no serious harm has yet been demonstrated.

Finally, we wish to make it clear that our experiments do not deal with the mechanism whereby intranasal medicaments in the human being may reach the lungs. All they demonstrate is, that when such materials get into the lungs of healthy rabbits, they may be extremely toxic and harmful. The possibility remains that if such materials get into human lungs, they may cause similar harm; their use intranasally, therefore, should be attended with a realization of their potential dangers.

SUMMARY AND CONCLUSIONS

Various aspects of the problem of intranasal medication have been considered, both from the clinical and experimental points of view. From a review of the literature the following conclusions appear warranted: The most suitable vehicle for intranasal medicaments is isotonic salt solution, inasmuch as it causes no demonstrable interference with ciliary activity, whereas both hypotonic and hypertonic solutions cause definite harm to cilia. Oils, as vehicles, are unsuitable not only because they interfere with normal streaming of mucus, but also because, being separated from the epithelium by the blanket of mucus, the drugs dissolved in them act less effectively.

Drugs are used in the nose for their antiseptic, astringent or vasoconstrictive effect. Of the vasoconstrictors, only weak saline solutions of ephedrine have so far been definitely shown to cause no damage to ciliary activity or mucus flow. The astringents, besides causing anosmia, may cause immediate cessation of ciliary action. Although some of the so-called antiseptics, viz., mild silver proteins, cause no apparent harm to ciliary action, their antiseptic value is doubtful, in view of our observation that the bacteria in bronchiectatic pus grew freely for as long as 44 hours after mixing the pus with equal parts of solutions of these drugs.

A review of the literature reveals the rapid increase in incidence of lipoid pneumonia following intranasal use of oils, thus constituting a natural experiment proving the ease with which such oils may go directly from the nose to the lungs. We report three cases in adults of such lipoid aspiration.

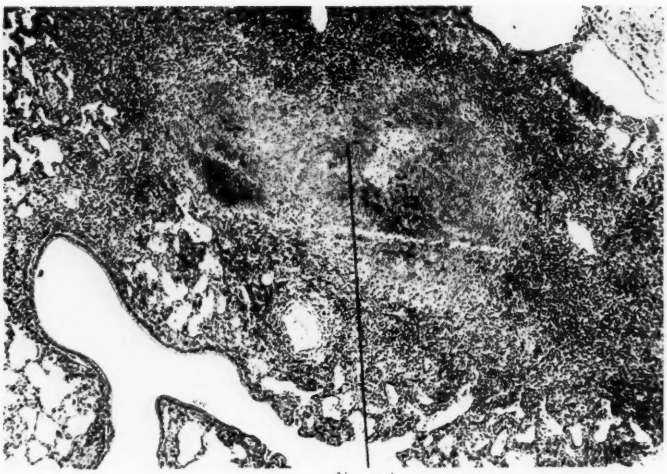


Fig. 21. Photomicrograph of a lung of a rabbit that received 0.5 cc. of 1 per cent tannic acid in each nostril once. Sacrificed two days after the treatment. Note the peribronchial necrosis. x80

In view of the fact that necropsy studies of such conditions deal only with end-stages, we have studied the early changes in the lungs of normal rabbits, following intranasal instillation of oils and watery solutions of antiseptics, astringents or vasoconstrictors. The general findings were as follows:

1. Oily solutions, when instilled intranasally in small amounts into healthy rabbits, went quickly to the lungs and caused edema, desquamative alveolitis and focal lipoid pneumonia. When mixed with living bacteria, cultured from the nose of a rabbit with snuffles, they caused, after two to three weeks, granulomatous lesions containing oil.

2. Watery solutions of antiseptics and astringents similarly entered the lungs and caused, not only edema, but severe focal necrosis, purulent bronchitis and bronchopneumonia. This was particularly true of the antiseptics when given intranasally to rabbits with snuffles or when given mixed with living micro-organisms.

3. Isotonic saline solutions of such vasoconstrictors as ephedrine and neosynephrine, caused no significant degree of pulmonary damage after intranasal instillation in normal rabbits.

4. The potential dangers of intranasal medications are discussed. The conclusion is that the only completely safe medicaments are weak saline solutions of appropriate vasoconstrictors.

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XLVI

AN EXPLANATION OF THE RESPIRATORY FAILURE SOMETIMES OCCURRING AFTER A SUCCESSFUL TRACHEOTOMY*

V. E. Negus, M.S., F.R.C.S.

London

Some years ago Sir St. Clair Thomson directed my attention to the peculiar fact, observed by him, that some patients, after complete relief of laryngeal obstruction by tracheotomy, might cease breathing for no obvious reason and even die. A tour he had made of all the Museums of the Medical Schools of London revealed a considerable number of specimens of laryngeal stenosis, from various causes, in which it was patent that this had been relieved by a correct and perfectly successful tracheotomy, and yet the clinical report recorded that the patient, with a completely restored airway, had succumbed to heart failure, with symptoms of embarrassed breathing.

His reference was, of course, to obstructions of some standing and not to acute conditions, such as diphtheria, where many factors interact to produce serious symptoms.

It appeared necessary to consult physiologists, and particularly those with especial knowledge of respiration and biochemistry. The answer to the problem was supplied after personal inquiries to Professor R. J. S. McDowall. Investigations on patients were made by Dr. R. D. Lawrence, who gave me the information sought for. The writings of Professor J. S. Haldane and Dr. Yandell Henderson were consulted, as therein is contained all the necessary information on gaseous exchanges in the lungs. Dr. McCance has kindly criticised and corrected the conclusions reached and has also investigated certain of the patients.

The respiratory center depends normally upon the concentration of hydrogen ions in the blood reaching it and this in turn depends upon the functional activity of the lungs; for carbon dioxide regulates the pH of the blood and is removed via the lungs. The

*Read before the American Laryngological Association, by invitation, at the meeting held in Atlantic City, May, 1938.

larynx normally takes an important part in regulating the respiratory exchanges.

The blood is weakly alkaline in reaction, its pH being 7.4; any increase or decrease of the free hydrogen ions would alter this reaction, but in point of fact it is of remarkable constancy. The reaction depends upon the ratio of CO_2 present as carbonic acid, to that combined with sodium as bicarbonate.

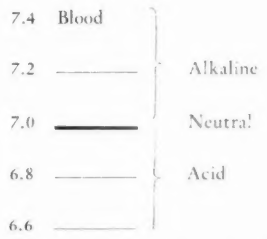

Fig. 1. Reaction of the blood.

The percentage of CO_2 in the arterial blood is constant in health and is normally 5.6; that in the venous blood is about 8.0 per cent. In diseased stages the percentages may be higher, as will be mentioned later. The CO_2 in the arterial blood is in equilibrium with that in the alveoli of the lungs and it follows, therefore, that as the percentage in the blood must be kept constant, so also must that in the alveolar air remain at 5.6 per cent; the tissues generally, and the respiratory center in particular, are most intolerant of any change in the reaction of the fluid bathing them. In normal health any excess of acid is removed by increased lung ventilation; the individual breathes more deeply, rather than more rapidly, and washes out an increased quantity of CO_2 from the alveoli, thus keeping the arterial content at 5.6 per cent, with maintenance of a normal acid base balance.

The larynx has a considerable regulating influence on these exchanges: the glottis widens on inspiration and becomes narrower on expiration, thus exercising some control over both inspiratory and expiratory currents of air. These glottic movements affect the Hering Breuer reflex, which determines the rate of respiration; the slowing effect gives time for CO_2 to be removed from the blood and oxygen to be taken up. There is, at the same time, considerable influence on the rate of blood flow through the lungs, dependent upon pump action, with dilatation of the pulmonary capillaries and

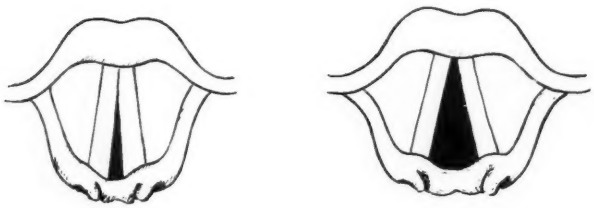

Fig. 2. Exaggerated respiratory movements at the glottis.

suction action on the right auricle and great veins during inspiration. The controlling mechanism is quite efficient in normal health and is extremely sensitive; a rise of 0.2 per cent of CO_2 in the alveolar air produces an increase of 100 per cent in the lung ventilation. Alterations in the oxygen content of the blood, on the other hand, have but slight effect on the respiratory center.

Now, if the larynx becomes obstructed for some reason, this normal mechanism is brought into greater activity and the depth of respiration is increased; this entails considerable effort, as is required in laryngeal stenosis, and may be beyond the powers of the patients to sustain. Furthermore, the forced inspirations, against obstruction, cause the blood stream to rush through the lungs without sufficient time for the necessary respiratory exchanges. This occurs particularly in patients suffering from double abductor paralysis. Some other means must, therefore, be found to maintain the normal reaction of the blood. The changing factor is the alveolar CO_2 content, and consequently the CO_2 percentage in the arterial blood. In certain cases of laryngeal obstruction investigated for me by Dr. Lawrence, this rose to percentages of 6.2, 6.4, 6.6, 6.8, 7.0, 7.2 and 8.0; these figures show an enormous increase and yet they were well neutralized by some new mechanism. The latter is the withdrawal of sodium from combination with the proteins and other buffer substances and recombination with carbonic acid to form sodium bicarbonate; this maintains the constancy of the acid alkali ratio. The sodium bicarbonate in the plasma may be estimated by biochemical methods and the findings can give a close indication of the alveolar CO_2; the latter is difficult to estimate accurately in patients suffering from laryngeal obstruction. The increased output of alkali combines with the rising content of carbon dioxide and prevents any

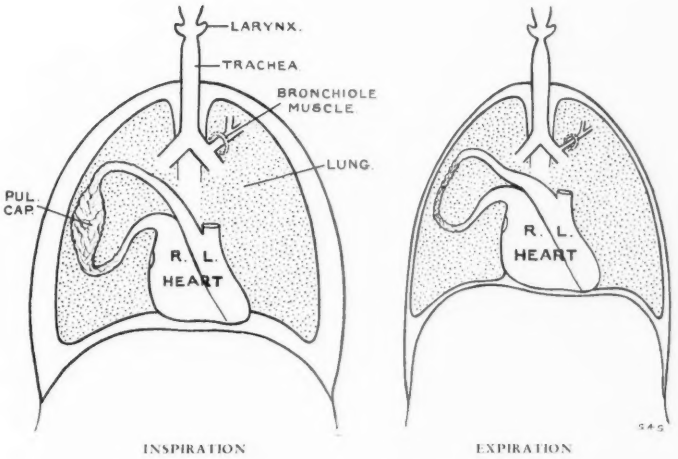

Fig. 3. Respiratory changes in the pulmonary vessels.

At full *inspiration* pressure in the thorax is at its lowest level. The glottis is well open, the bronchial muscle is relaxed and air enters the alveoli freely. Relief of pressure from the walls of the pulmonary capillaries causes them to dilate; blood is thus allowed to rush freely from the well-filled right side of the heart, through the lungs to the left ventricle, whence it is pumped out at high pressure.

During *expiration* the pressure rises and the pulmonary capillaries are compressed. Because of increased pressure on the great veins and right side of the heart, the flow of venous blood is impeded. Circulation through the lungs is slowed down and the left side of the heart receives less blood than during inspiration. Aortic pressure therefore falls. The bronchial musculature is in contraction and the glottis is less widely open than during inspiration. Escape of air from the lungs is thus obstructed.

excessive increase in the percentage present as carbonic acid. The reaction of the arterial blood is thus maintained at a pH of 7.4, without any immediate exhausting call on the muscles of respiration. The respiratory center is now set at a higher level, and responds to blood containing more than the normal percentage of CO_2. Some of the increased acid is eliminated by the kidneys, chiefly as phosphates. During these changes there may have been no anoxaemia for the reason that there is, in normal conditions, a reservoir or excess of oxygen in the lungs; the patient is, therefore, not cyanosed if the obstruction has been of gradual onset, unless it reaches an extreme degree.

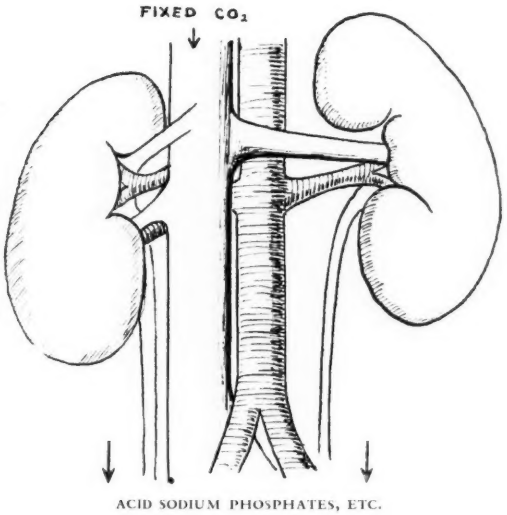

Fig. 4. Removal of acid from the body. Diagram to illustrate elimination of CO_2, combined as acid sodium phosphate, etc.

But there comes a time when increasing obstruction prevents sufficient oxygen from reaching the alveoli, while the rapidly circulating blood has not had sufficient time to take up what oxygen is present. The distress of the patient then calls urgently for tracheotomy. Forewarning of the necessity for this operation could be provided by estimation of the alkali reserve, as follows from the observations made above.

The tracheotomy may proceed in a tranquil manner. The cannula inserted into the trachea at once gives free passage for entrance and exit of air. There has probably been no strain or coughing if a small quantity of cocain has been injected into the lumen of the trachea before opening it.

Breathing immediately becomes deeper and also somewhat faster, owing to removal of obstruction, and acceleration of the Hering Breuer reflex. Lung ventilation is suddenly and enormously increased and much CO_2 is washed out of the alveoli and blood. In one case of mine, examined by Dr. Lawrence, the percentage of alveolar CO_2, and, therefore, of arterial CO_2, was brought back from

7.2 per cent to the normal 5.6 per cent after a few breaths and within so short a time as five minutes. But still, in spite of the abundant and clear airway, respiration may cease immediately, or after an interval of as much as one hour, just because of lack of stimulation of the respiratory center. The condition is quite similar to that following voluntary over-breathing or what we can observe after forced artificial respiration in an experimental animal. For the time being the acid base ratio is upset; the percentage of free CO_2 present as carbonic acid is suddenly lowered, while that fixed as bicarbonate remains stationary; the arterial blood has become so alkaline that the respiratory center no longer receives the necessary stimulation. Respiration then ceases because of acapnia. Oxygen lack is soon felt, but may have little effect on the respiratory center, which is bathed by such alkaline blood that it ceases to respond to even the extremest stimulus of want of oxygen (Haldane). The patient may die without ever taking another breath. This time of danger, during which excess of alkalies is fixing too much of the available CO_2, may extend over some hours.

The practical deductions of all this are obvious and are probably utilized by many surgeons, although the rationale may not be thoroughly understood.

At the time of operation on any cases of laryngeal stenosis a cylinder of CO_2 should always be at hand; and if breathing becomes shallow or of Cheyne Stokes type, the patient should be made to inhale the gas. This respiratory stimulant should remain at the patient's bedside for some hours, by which time the balance will have been readjusted. Administration of ammonium chloride before operation could be adopted, but is less simple and less adjustable than the method described.

When the problem is considered it really becomes a simple one, with an obvious remedy, but neglect of the necessary precautions may lead to grave disaster.

One other practical point has already been referred to, i.e., the advisability of estimating the alkali content in cases if there is doubt as to the degree of obstruction. But clinical care should be sufficient. Every patient with sufficient laryngeal or tracheal stenosis to cause any audible stridor on exertion should be advised that he is in a danger zone. If the stridor is noticeable when he is resting, the question of tracheotomy should be considered. If it is audible when he is asleep, the operation is urgently required. As we perform it nowadays a tracheotomy should be a straightforward performance

without risk. A recognition of the possibility of this condition of acapnia and of the means of overcoming it, should remove the possibility of anxiety in the after-care of such cases.

NOTE:—I am much indebted to Professor McDowall, Dr. Lawrence and Dr. McCance for their advice and practical help. I am also particularly grateful to Sir St. Clair Thomson, not only for initiating this inquiry, but also for reading, correcting and making additions to this paper. His opinion on the indications for tracheotomy has been of particular value.

XLVII

PSYCHIATRIC THERAPY IN DYSPHEMIA AND DYSPHONIA: STUTTERING, PSYCHOPHONASTHENIA, APHONIA, FALSETTO*

JAMES SONNETT GREENE, M.D.†

NEW YORK CITY

The speech disorders which I shall presently consider, the so-called functional disorders, are presumably of psychic origin with no organic involvement. However, we have come to the realization that, biologically, we can never classify a disorder as purely functional or purely organic; it is always a question of considering the organism as a whole.

The functioning of the human organism as a finely co-ordinated, rhythmic, adjusted unit, depends on that internal physiologic pattern or configuration, found in all of us, which regulates and co-ordinates the reactions of the individual as a whole. This means that theoretically, in the so-called normal individual, there is perfect synchronization of the physical, mental and emotional phases of his makeup.

The general assumption is that the mental, physical and emotional aspects of the individual show a more or less equally progressive rate of development. Usually, however, this is not so. Due to heredity or some other predisposing factor, the physical may outdistance the mental; the mental the physical; or, as is more frequently observed, both the physical and mental may outdistance the emotional. It is therefore just as important to know how mature a person is emotionally as to know how far he has advanced physically or mentally.

This is particularly true when we consider that an innate tendency toward emotional and nervous instability underlies the neuroses, and that, to a greater or lesser degree, the dormant seeds of a neurosis are present in all of us. That is, we are all liable to peculiar states of tension or irritability of the nervous system.

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†Medical Director, National Hospital for Speech Disorders, New York City.

These states, which underlie the neuroses, are generally not regarded as organic, but are considered as behavior states which seemingly affect only a part of the personality of a nervous (or neurotic) individual. The general idea that these conditions have a nonphysical basis is predicated upon the absence of any evident lesions. Our observations, however, definitely point to the fact that some individuals show marked evidence of an inherited neuropathic diathesis and that, regardless of the absence of any evident lesions, the abnormal behavior of such individuals depends on defects, limitations or faulty action of their anatomical mechanism.

Neuroses, then, should never be considered apart from the organism, but as an integral phase of that organism.

A neurosis as it develops may take on various definite forms, but the commonest of all are the anxiety states in which the person is continually in a state of fear, expressed both physically and mentally. This fear, which is termed neurotic anxiety, is perceived psychically as an unknown danger threatening the personality. When the fear takes a definite form, it is a phobia. For example, an individual may suffer from a fear of enclosed spaces (claustrophobia), or open spaces (agorophobia), loud talking (phonophobia), speaking (lalophobia), and so on.

Those suffering from the voice and speech disorders I am about to consider all demonstrate neurotic anxiety of one form or another. Our long experience has led us to the conclusion that all these patients fall under one general classification—they are neurotics of the sympathicotonic type. Although their respective clinical pictures vary in the number, character and intensity of the presenting symptoms, certain subjective and objective symptoms are common to all of them.

Physically, patients who suffer from the so-called functional speech disorders are characterized by a constant state of nervous tension. They are tense, restless, apprehensive and uneasy, and they show a marked lability of the pulse and blood pressure, with unusuai tachycardia when under emotional stress. They generally are afflicted with hyperhidrosis and have cold, clammy hands and feet. While their basic metabolic rate is normal or only slightly below normal, they demonstrate a tendency toward marked fatigue.

Further, these patients possess a relatively high potential for the spread of emotional tone (that is, a tendency for dominance of the sympathetic division of the vegetative nervous system) which far overreaches that of the normal individual.

Anxiety neuroses and phobias of various kinds are common, and neuroticism is usually found to be a familial trait. In the words of Dr. Stanley Cobb,[1] these persons are "born tender, and are exquisitely vulnerable to the hurts of the world."

In temperament they are generally absorbed with themselves and self-centered (narcisstic). In their contacts with the external world they are rather aloof, subjective, and egocentric—all of these traits usually being defense mechanisms against basic anxiety and feelings of inadequacy or inferiority.

Very often an individual possessing a neurotic personality of this type, in order to escape from what is to him an intolerable situation, will convert his unconscious neurotic anxiety into a physical manifestation. Quite frequently this conversion process is localized in the vocal tract and the patient will present many very real symptoms of vocal tract involvement. In other words, while the etiology or the more immediate origin of the symptomatology is psychologic, the expression is physiologic. This is seen in dysphemia (stuttering) and in the various form of dysphonia, such as psychophonasthenia, hysterical aphonia, and many cases of falsetto voice.

We will now consider the first of these conditions—stuttering.

DYSPHEMIA (STUTTERING)

Stutterers in general do not realize that they are neurotics—and, furthermore, neurotics of a special type. They are sympathicotonic neurotics whose disorganization, both psychic and somatic, is so marked and so common to all of them that they can be classed as "stutter-type neurotics."[2]

The neuropathic diathesis of the stutter-type neurotic is an hereditary trait. In these people there is apparently a disorder passed on from parents to children which predisposes them to nervous and emotional instability, but which is only to a degree a specific organ weakness or inferiority. The chief factor seems to be a disturbance in some stabilizing mechanism—probably a hypothalamic involvement.

Since the hypothalamus is in direct control of the vegetative nervous system, and therefore in direct control of the bodily manifestations of the emotions, an involvement in that organ would account for the stutterer's relatively high potential for the spread of emotional tone, and for his vegetative imbalance as manifested through the sympathetic symptoms mentioned before.

This physiologic idea is substantiated by Paul Schilder's[3] implications regarding stuttering, which also bear out the fact that there must be a stutter-type. He points out that there is a correlation between stuttering and tics, so that we can refer to both as "striate body neuroses."

Further, the stutter-type belongs to that group of individuals which Professor Pavlov[4, 5] classified, in terms of nervous excitation and interruption of psychomotor function, as "strong but unbalanced." In this group there appears to be a definite clash between the excitatory and inhibitory functions of the brain cells, so that the stutterer demonstrates a combination of the harmful effects of excessive excitation and abnormal interruption in his motor functions.

In other words, there is an interference with that inner rhythm so necessary for the integrated functioning of the individual. Rhythm, as we know, is an innate characteristic of the human organism. It has its beginning in the cell, and is a general characteristic of the organism as a whole—an integral phase of all our psychic and somatic activities.

The stutterer's lack of rhythm is fundamental, and when we consider that his chronic tendency toward arrhythmic response is combined with the high emotional tone of sympathicotonia, we can readily see why he is predisposed to disorganization and chronic hesitation in general.

Because of this predisposition to chronic hesitation in general, the stutter-type person may be said to stutter in more ways than in speech. We have treated people who stuttered playing musical instruments, some of them giving a spasmodic repetition of notes on the violin, and others becoming stuck on a note on the piano. We also have had patients who make the typewriter or linotype machine stutter, and others who demonstrate their hesitations in such acts as dancing, golfing, and driving. (Almost everyone has seen stuttering automobiles.)

This general disorganization in the stutter-type is due to the fact that balance between his mental, emotional and physical states is disturbed. His organic motivation is never stable. There is always an abnormal striving for dominance between his emotional and intellectual states, with the emotional element almost always predominating, so that reactions are out of all proportion to the strength of the stimuli.

The forces of stimuli as interpreted by his neuropathic organism come into overpowering conflict with his desire to hold his balance of intellectual ascendancy. Quite often his emotions cloud his intellect to such a degree that his whole mental state is one of turmoil. Consequently, under stress, behavior in the stutter-type takes the form of irregular, hesitating physical manifestations, which may be apparent in any of his psychomotor activities. When speech is involved we designate it stuttering.

Let me emphasize the fact that, even with his neuropathic diathesis, the stutter-type individual is not necessarily destined to stuttering speech. Only an unfavorable environment—that is, an environment of opposition—which accentuates the native conflicts of the individual, can evoke stuttering in its various forms.

This is borne out by the fact that the adult stutterer usually gives a history of having been a nervous, fearful child, often living in a psychoneurotic parental atmosphere surcharged with nervous tension.

Almost any shock or strong environmental impact is enough to precipitate a speech disturbance in a child with this neuropathic background,[6] living in an oppositional environment; and such a child is almost certain to invest this initial speech disturbance with emotionalism.

The memory of the experience remains vividly with him, so that he carries over the idea of failure and humiliation and wonders if he will again have difficulty. Of course, in this negative state of mind the failure is repeated—he does have difficulty. This experience happens again and again, until a definite nerve pathway via the cerebral cortex is formed, and we have stuttering as a conditioned reflex.

As the child grows older and the social situations become more complex, a chain of humiliations, failures and frustrations is set up, resulting in an anxiety-fear neurosis which in time warps the entire personality. Due to his speech disorder, to his tendencies toward chronic hesitations in general, and to his many frustrations, feelings of inadequacy and inferiority soon develop and the stutterer becomes the passive, egocentric individual whom we have already described as a definite pathologic entity—the stutter-type.

His stuttering is not a speech defect as the term is conventionally understood, but a deficiency specialization, and all therapeutic measures instituted for stuttering speech must take into consideration the function of the organism as a whole—the entire personality.

PSYCHOPHONASTHENIA

A deviant of the stutter-type is the individual with normal verbalization but abnormal vocalization. The condition is often erroneously diagnosed as phonasthenia, or weak voice. The symptomatology is variable. The patient complains mainly of a tight, constricted throat, uncontrollable choking sensations when speaking, and a strained, weak voice without carrying power. To the listener his voice presents other marked characteristics. It is strident, rasping, grating; in fact, a croaking, pinched voice with pitch irregularities.

The condition has been variously attributed to a forced technique, as over-fatigue in singers and speakers; to local involvements of the organs of speech; and to physical or mental sufferings. While there may be an element of any one of these factors present, none of them satisfactorily explains the basic or underlying cause of the condition.

For, although the patient always complains of unusual effort and fatigue, there is no causal fatigue laryngologically, no local involvement, no physical illness; but there is a decided causal psychic state. These patients, like the stutterer, suffer from an anxiety neurosis. Their anxiety states cause misdirected laryngeal effort to which, in the course of time, they become conditioned. The associated muscle groups become strained and uncertain, and a more or less chronic state of fatiguability results, which is often mistaken for the causal factor in the voice condition.

Since this seemingly is a throat condition, the patient in the majority of cases visitis the laryngologist in the hope that his throat will be relieved of its "weakness" so that he may again speak clearly and distinctly. Invariably, the patient is told that there is nothing markedly wrong with his throat. The slight lack of approximation of the vocal cords or irregular contact of the cords, or even a slight congestion which is often noticed, is usually not enough cause for the chronic strained voice condition.

The reason for the general negative laryngological findings is simply that the condition is not dependent on local pathology, but is due to psychic factors that have their roots in the atypical constitutional makeup of the individual and his atypical psychological development. In spite of the laryngological syndrome, the condition is essentially a psychobiologic one. The vocal manifestation, like the speech disturbance of the stutterer, is merely a symptom of the individual's neurotic anxiety. Since the problem is fundamentally

psychic—emotional—we at our clinic have come to designate this condition as psychophonasthenia.

Paradoxical as it may seem, these patients do not suffer from a weak larynx, or from functional voice fatigue, as pointed out by Flatau,[7] Froeschels,[8] and others, but from a larynx which in reality is over-powerful. The patient uses his larynx as an instrument of pressure instead of an instrument of performance. He inhibits voice production through muscular pressure, vocalizing *with* his throat instead of *through* his throat.

The psychophonasthenic's method of vocalization diminishes the carrying power of his voice. Exaggerated pressure causes the thyroid cartilage to be drawn up to the hyoid bone, so that the arytenoids are rotated on their facets, articulating them with the cricoid cartilage. Thus the vocal cords are adducted to a tighter contact and in their movements create greater friction, which results in a tense form of phonation instead of a normal resonant voice.

All this is merely a physiologizing of the emotions. Due to his constitutional makeup and his atypical psychological development, the indivdual acquires an unconscious fear or feeling of inadequacy —a fancied or real inability to measure up to a necessary standard in a competitive world—which in the course of time results in a neurosis.

The patient's extreme laryngeal manifestations may be said to be a deficiency specialization—an hysterical mechanism which he uses as a defense measure. This is borne out by the fact that the condition is usually developed during late adolescense or adulthood, and is most often observed in those whose vocation especially calls for public contacts—ministers, lawyers, singers, and many others.

Since basically psychophonasthenia and stuttering depend upon the constitutional makeup of the individual and his atypical psychological development, a comparative table of the two conditions should prove interesting:

PSYCHOPHONASTHENIA AND STUTTERING

A Comparative Table

PSYCHOPHONASTHENIA	STUTTERING
TYPE OF NEUROSIS	
Transference Neurosis. Is more capable of objective relationships; invests more emotional interest in others.	Narcisstic Neurosis: Stutter-type. Objective relationships less mature; emotional interest invested chiefly in the "self."
ONSET	
Adolescence and Adulthood.	90% preadolescence.

Course	
Chronic or episodic.	Chronic.
Outcome	
Spontaneous resolution uncommon. Of Cases Treated: Various degrees of improvement.	Spontaneous resolution during childhood, 10 per cent. Of Cases Treated: 70% recovered. 20% much improved. 10% improved.
Etiology	
Familial Predisposition: Not prominent. Psychological Organization: Adolescent. Essential Mechanism: One of making mature adjustments. Voice Symptom: Crystallizes his compromise solution in adult relationships. Use of Symptom: To mask selective incapacities. Protection for a number of complexes in an otherwise mature organization.	Familial Predisposition: Marked. Psychological Organization: Infantile. Essential Mechanism: Flight from mature adjustments. Speech Symptom (Stuttering): Crystallizes his infantilism in adult relationships. Use of Symptom: Protection for his infantile complexes which are part of his immature organization.
Symptomatology	
1. Type of Neurosis: Hysteria. 2. Type of Symptom: Somatic conversion. 3. Symptoms rejected. Regards condition as foreign, unnatural and not utilizable. 4. Tone production affected. (Vowels)? 5. Whispering: unimpaired. 6. Voice and Speech at high level: usually not involved. 7. Shouting: impaired. 8. Singing: as difficult as speech. 9. Episodes or intervals of perfect speech: infrequent. 10. Frequently normal speech for short time on awakening. 11. Environmental influences modify voice and speech only slightly. 12. When alone: slight improvement. 13. Awareness of laryngeal and abdominal constrictions. 14. Social anxiety: slight.	1. Type of Neurosis: Compulsion. 2. Type of Symptom: Repetition-compulsion (tic). 3. Symptoms accepted. Regards condition as part of organic whole, natural and utilizable. 4. Speech production affected. (Consonants)? 5. Whispering: usually not impaired. 6. Voice and Speech at high level: usually not involved. 7. Shouting: not impaired. 8. Singing: not difficult. 9. Episodes or intervals of perfect speech: frequent. 10. Frequently worse on awakening. Characteristic sluggishness on changing from sleeping to waking states. 11. Environmental influences modify speech markedly. 12. When alone: marked improvement or normal. 13. Often no specific awareness. 14. Social anxiety: decidedly marked.

CHARACTER TRAITS

Total Behavior and Thought Patterns

General Behavior:	General Behavior:
More direct, definite and decisive.	Vague, circumstantial and diffuse.
Traits:	Traits:
Free; flexible.	Compulsive; rigid.
Expression:	Expression:
Emotional.	Intellectual or theoretical.
Group Response:	Group Response:
Asocial but not retiring. Usually no difficulty in joining group but keeps aloof.	Social but retiring. Has difficulty in joining group. When he does, is not aloof.
Conflict.	Conflict:
Positive and negative mental impulses irrespective of environment.	Positive and negative mental impulses precipitated by social contact.
Personality:	Personality:
Much more mature; more efficient; does not hesitate.	Much less mature; less efficient; chronic hesitator.

In summary, we may say that the psychophonasthenic suffers from a conversion neurosis. His personality structure is much more mature and flexible than that of the stutterer, and his voice disorder is an unconscious compromise which legitimizes his withdrawal from social contact.

In the stutterer, on the other hand, we are dealing with a character neurosis, one in which fundamental personality traits have been arrested in their development. This arrest takes place early in childhood and is maintained, although completely unconscious to the patient, through anxiety and fear. The stuttering symptom exemplifies the conflict between these two aspects of the personality, but primarily serves the infantile one.

In both psychophonasthenia and stuttering the symptom is maintained as long as its unconscious roots remain intact.

HYSTERICAL APHONIA

We will now consider another type of patient whose cardinal symptom is also of a misleading nature—the patient who suffers from hysterical aphonia, or functional loss of voice. The physical dysfunction in this disorder, like that of stuttering and psychophonasthenia, is due to neurotic background and psychic influence; but the onset, course and nature of aphonia differ markedly from the onset, course and nature of stuttering and psychophonasthenia.

In hysterical aphonia, the onset of the disorder is relatively sudden and acute, rather than gradual and cumulative. Of course, since

the individual suffering from this disorder is also a neurotic of the sympathicotonic type, there has been a long-growing anxiety-fear neurosis. His neuroticism, however, does not become fully apparent until the individual suddenly displays physical symptoms—loss of voice.

In the cases that come under our observation, the patient almost invariably has lost his voice following a "cold," so that the organic plausibility of the symptom is extraordinarily thorough. Not only is the patient saved from a conscious realization of the true nature of his disorder—that it is an escape mechanism—but in many cases the disguise is so perfect that even the medical consultant is deceived.

Because the peripheral manifestation of this disorder is in the vocal tract, these patients usually consult the nose and throat specialist. Confronted with this type of patient, although finding no signs of paralysis or any other special condition of the vocal tract, the laryngologist naturally resorts to local physical therapeutic measures as a matter of routine.

Sometimes in an effort to produce normal voice or speech in the patient, his routine therapy may take a rather strenuous form. This type of procedure may or may not aid the patient. In fact, it has been found to be quite detrimental in many cases. The beneficial results of shock therapy are almost always transitory, because frightening a patient into vocalization does not counteract his basic neuroticism.

If the cause of the aphonia is not brought to the surface and to the conscious realization of the patient, the therapist will have difficulty in alleviating the condition; and if the voice is restored, a relapse will usually follow. The anamneses of many such patients reveal that in practically every case where local therapeutic measures have brought relief, recurrences have taken place.

In the cases of hysterical aphonia that come under our observation, the initial procedure is to compile as complete an anamnesis as possible. When this is done, one usually finds that the voice condition is just a vocal projection which masks the patient's underlying difficulty—it is a neurotic escape from an environment which is distasteful or which threatens his or her security or happiness.

Since this is so, and since, with minor exceptions, the laryngological findings are negative, it stands to reason that all physical therapy must be secondary. Initial treatment should always be on the nonactive side. I do not mean to imply that the laryngologist

should immediately refer the aphonia patient to the psychiatrist for treatment.

Indeed, it is a principle in psychiatry that it is impossible to undertake the usual orthodox measures during acute stages of certain conditions. Consequently, the point I wish to make is that in these aphonia cases, a simple and practical psychiatric procedure which can readily be mastered is in place, and the laryngologist himself must be prepared, as a matter of routine, to institute it before resorting to any other means.

A brief resume of a few cases will illustrate the nature of this disorder and the type of procedure I have in mind:

REPORT OF CASES

CASE 1.—J. G., married woman, age 30. Recurrent loss of voice.

Patient first lost her voice after an attack of tonsillitis. Periods of aphonia alternated with periods of normal speech for the next four years. During that time a tonsillectomy was performed, a number of examinations were made, x-rays taken to allay suspicion of tuberculosis; she had throat and sinus treatments, and a series of injections, but all with temporary results.

Six months ago the case came under our observation. Complete anamnesis revealed that the patient unconsciously resented the affection her husband bestowed on their children. It was a blow to her narcissism. Her unconscious solution was to gain his attention by the physiologizing of her emotions through a conversion mechanism.

Through psychiatric counsel and suggestion, the patient was persuaded to accept the true meaning of her symptom. Her aphonia then disappeared and she has talked normally ever since.

However, I have since learned that our patient's conversion has taken a new form. She now believes she is pregnant. Despite such very positive symptoms as morning anorexia and a predilection for certain types of food, the obstetrician can find no evidence of pregnancy. Apparently her desire for attention still predominates and she has merely substituted one symptom constellation for another.

Technically, however, we can close the case with the terse statement, "patient cured of aphonia." She is definitely out of the realm of the laryngologist. He has been superceded by the obstetrician.

But is she cured?

CASE 2.—M. H., single woman, age 22. For six months not able to speak above a whisper.

Patient first lost her voice following a cold. She consulted a number of doctors, had her throat scraped and painted, had massage treatments, and underwent a tonsillectomy. Her condition did not improve.

A constant failure of glottal closure, with its resultant aspirate voice, gave her speech a "stage whisper" quality.

The patient displayed no apparent anxiety regarding her condition, and was surprisingly complacent about it. Examination revealed that the patient suffered

from myopia, and was extremely sensitive about her sight, for she was practically helpless without her glasses. She did not accept her visual defect, with its inconveniences and social handicaps, on an adult level; but unconsciously resorted to social withdrawal or flight, for which her loss of voice supplied the legitimizing sanction. She, therefore, displayed no genuine interest in treatment which threatened to remove her distraction and prevent her flight from reality.

CASE 3.—M. G., married woman, age 49. Recurrent loss of voice for past two years.

The patient, an active business woman in her husband's department store, suddenly lost her voice after returning with her husband from a buying trip abroad.

The findings of several physicians were negative, and a change of climate was advised. She came to New York and received a variety of treatments, including the extraction of several teeth, but her aphonia still persisted. Another change of climate was next advised, so she and her husband went on a cruise; but her voice condition was not influenced. Then, seven months after losing her voice, while away from home, and about to greet her son on Easter morning, she suddenly began to talk normally.

Ten months later, on returning home from another trip, she again lost her voice. As on the previous occasion, this occurred on the day of her return. After the aphonia had persisted for two months, she again came to New York and was referred to our clinic.

Our psychiatric study revealed that the patient's voice condition was an unconscious protest against an unescapable situation. Her return to her old routine precipitated both attacks of aphonia.

Although she participated actively in her husband's business and protested that she enjoyed the work, it was evident that she unconsciously rebelled against being tied down to the store and also against her husband's transference of attention to it. Her aphonia was her means of escape and at the same time a means of gaining attention.

In our psychiatric session with the patient we gave her a simple explanation of the underlying nature of her voice symptom. She readily accepted it, so that subsequent laryngological and vocal measures in the nature of a placebo, brought about desired results.

FALSETTO VOICE

Still another symptom complex is paraphonia, or falsetto voice. The voice of the male afflicted with this condition is thin in quality, high, shrill and piping in tone. It sounds forced or unnatural and, as its name implies, is a false voice—a child's voice produced by an adult.

Unfortunately, a popular stigma is fallaciously attached to the person with a falsetto voice, because this type of voice is characteristic of the eunuch and eunuchoid.

Eunuchoids are persons who, without being castrated, entirely simulate in clinical manifestations the true eunuch. The condition

is due primarily to a developmental disturbance in the gonads. Since a characteristic of the eunuchoid is a high-pitched voice, the term "eunuchoid voice" has come to be used synonymously with falsetto voice. This misunderstanding is unfortunate for the individual possessing a falsetto voice, since in almost all cases the stigma of doubtful sexuality is entirely undeserved.

Like the other disorders we have considered here, falsetto voice is usually a purely functional condition, with no pathologic involvements of the vocal organs or of the organs of generation. In its initial states, the condition is simply a neurotic manifestation of excessive sensitivity at the pubescent period.

A sensitive boy who shows neurotic tendencies, and who in general is out of balance during his adolescent years, is more than normally embarrassed and shocked by his general systemic changes, voice included. The boy in unconscious anxiety and embarrassment regarding his new status of physiologic maturity, strives to remain on his childish level. He clings to his high-pitched shrill voice, and since mental imagery and auditory memories determine tone production, he cannot produce a low voice when he is thinking in terms of his old high voice.

Through constant use of his high voice, he causes a faulty coordination and misdirection of the phonatory muscles. This condition soon becomes permanent. There is an abnormal tug on the levator muscles so that the larynx is raised to an abnormally high position in the throat. Accompanying this there is an abnormal action of the intrinsic muscles of the larynx; the tension between the cricothyroid and the thyroarytenoid (vocalis) muscles is irregular, so that interference with the complete vibrations of the vocal cords takes place.

The cords become reduced to half their length, making a short string which always produces a high note. The false cords sag through diminished tension. The size of the ventricles of the larynx become reduced, thereby reverting to the original child state and producing a change in resonance.

The condition, if uncorrected, continues throughout the individual's life, and the psychic traumas sustained by the supersensitive, neurotically inclined male adult because of his high voice have far-reaching and serious effects. The following excerpts from case histories convey very clearly the marked social implications which falsetto voice has for the sufferer:

CASE 4.—B. H., male, age 28: "I have had trouble with my voice being high (like a woman's) since I was 15 or 16 years old. Two years ago I went to a throat specialist, who told me there was nothing physically wrong with me. I have always been nervous, but seem to be more so since my voice changed. People always tease me about my voice. I am very self-conscious and avoid other people as much as possible."

CASE 5.—A. R., male, age 45: "Ever since I have grown up I have felt embarrassed when in a group of men. Whenever I speak, they all look around at me and laugh. I was in the army in 1918, and there I suffered untold agony. The other soldiers laughed at me and would make remarks in a loud whisper as I passed. I have become so sensitive about my voice that I will not even read aloud to my little daughter. Sometimes I feel I am going crazy, and I believe I would be happy just to lie down and die."

These statements, as well as the results of our own observations, denote that, as in the other disorders considered, the laryngological findings in falsetto voice patients are also negative. For that reason, local physical measures in themselves are of little avail.

The status of the larynx is primarily a matter of position and not of pathology; but the abnormally high position of the larynx, and what it implies, is usually not apparent to the examiner, because the introduction of the laryngoscopic mirror causes a change in conformity and muscular action.

In these cases not only must special laryngological procedures be instituted, but, as in the aforementioned cases, the patient's personality difficulties must be recognized. In addition, his musical consciousness must be taken into consideration. Here again, one readily appreciates that it is a matter of considering the organism as a whole.

A realization of this concept should lead one to establish a composite form of treatment that takes cognizance of all these factors. Thus, if the patient is fortunate enough to find a special therapist—in other words, a musical psychiatric laryngologist—desired results can readily be obtained. Such a physician institutes a composite form of therapy which takes into consideration the individual's psychic state, laryngeal condition, and vocal abnormality. Under such therapy the patient's voice is lowered to the normal male range and he is then able to make a satisfactory adjustment.

PROGNOSIS

The main determinant in the prognosis in all these abnormalities is the degree of healthy personality that the patient presents.

Since the onset of stuttering is almost always in childhood (90% of cases before the age of ten), the stutterer has very little opportu-

nity to attain any degree of healthy emotional development. Regardless of this, the prognosis is good under favorable environment and a composite therapy.

The psychophonasthenic usually has a foundation of healthy emotional patterns up to the age of adolescence or even adulthood, hence he has the advantage of being able to react in a more socially adaptable manner. Still, in the majority of cases, the voice disorder is so deeply ingrained psychically, and the personality maladjustment so intertwined with environmental conditions which rarely can be changed, that prognosis is none too favorable.

The person with falsetto voice, up to the age of puberty is a so-called normal individual. His neuroticism, which develops later and which in many instances is very severe, can usually be resolved when the voice condition is corrected. Consequently, the prognosis is very favorable.

In contrast with the other disorders here described, the circumstances which cause the onset of hysterical aphonia are situational, and the vocal manifestations are acute rather than chronic. If the therapist gains the full confidence and co-operation of the aphonic patient, so that either the precipitating environment or the patient's outlook on the environment can be changed, prognosis is most favorable.

TREATMENT

While treating all of these patients the therapist must realize that the individual's abnormal psychology is based on a physiological foundation. That his thoughts, feelings and actions are governed by the defects and limitations of his anatomical mechanism. That although his condition shows no evidence of organic lesion, still there exists a form of physical inferiority which influences the organism as a whole.

Treatment of dysphemia and psychophonasthenia, due to the severity and chronicity of these two conditions, must be of a highly specialized nature extending over a long period. Hysterical aphonia and falsetto voice require a relatively shorter and less fundamental therapy.

The stutterer's neurosis is a social or "contact" neurosis. Because of his substratum of faulty personality integration, he has not been able to withstand the pressure of the everyday world. Therefore, in the stutterer the main goal of treatment is the development of character—the development of an integrated and more mature personality.

Therapeutic measures to be effective must give the stutterer an opportunity to try himself out in a specially created environment. Such an environment effects a rapprochement toward reality; it bridges the gap psychologically between the stutterer and the outside world. It reorganizes his entire life pattern, so that he faces the future unhesitatingly and with courage.

This sort of therapy is definitely out of the realm of the laryngologist, because the stutterer must receive a composite treatment of a special nature which includes both individual and group psychiatric therapy. After having treated more than 20,000 of these cases, I have come to the conclusion that a medical-social clinic is the only successful medium for carrying out such therapy.

The therapeutic measures as carried out at our medical-social clinic[9] have demonstrated that there are tremendous developmental potentialities in the stutter-type individual. Through our group psychotherapy, which we term "open-door psychiatry," and through individual counsel and persuasion, the stutterer learns to acquire emotional stability so that he is capable of taking an objective, nonhesitating stand and facing situations squarely on an adult level of reality. This rehabilitation is accomplished through a composite therapy of a medical, psychiatric, re-educational and social nature.

In psychophonasthenia, just as in stuttering, the laryngologist or the general practitioner alone cannot administer adequate therapy. Since the basis of this disorder is on a deep level, psychiatric collaboration is most advisable. At the same time, a course of special training should be introduced to counteract vocal anomalies brought about through misuse of the larynx.

I have already mentioned that in the case of the falsetto voice patient, the laryngologist must have both psychiatric and musical leanings in order to bring about a complete rehabilitation of the individual.

The same general therapeutic idea applies to hysterical aphonia; but here, as was indicated by the case histories cited, there must be a more thorough psychiatric therapy.

In conclusion, let me again emphasize the fact that for all of these conditions therapy must be of both a local and a general nature. It is of the utmost importance that the therapist consider the presenting physical symptoms as secondary defense mechanisms, and not as the principal object of treatment. Local therapeutic measures in these

cases must always be preceded by a brief course of practical psychiatric investigation and suggestion.

A careful anamnesis will generally uncover the underlying factors that have caused the patient to resort to physical symptoms. The laryngologist, provided he possesses the right kind of a personality, can readily administer a modified form of practical psychiatric therapy.

Psychiatric measures should be directed toward bringing the patient's unconscious motives into the consciousness. When this is accomplished, fundamental personality changes occur. The patient then accepts himself as he is, making the most of his physiological foundation.

126 East 30th Street.

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XLVIII

INCIDENCE OF STUTTERING AMONG THE DEAF

OLLIE BACKUS, PH.D.

SLIPPERY ROCK, PA.

It has been widely assumed that deaf people do not stutter. Explanations for such a hypothesis, coming from teachers of the deaf and teachers of speech correction, may be summarized as follows: (1) The speech of the deaf is scanning and stuttering does not occur in scanning speech; (2) speech is not used spontaneously for communication by deaf people, hence they are not under the same social pressure; (3) individuals deprived of hearing are subjected to less stimuli from the environment, hence are not as nervous; (4) deaf children acquire speech, not in the usual haphazard fashion, but by trained teachers of speech correction; (5) because of the inevitable language handicap, deaf children suffer less from conflict of thinking faster than they are able to speak. This information was volunteered in answer to questionnaires, mentioned farther on in this article.

Voelker[1] has reviewed the literature on this subject and has reported one case of stuttering in a totally and congenitally deaf boy. It is the purpose of this article to establish further the fact that deaf people do stutter.

Questionnaires were sent to all residential, denominational, private and day schools for the deaf in this country.[2] The first question asked was: "Do you know (or have you ever heard of) a totally or partially deaf person who stutters now or has ever shown signs of stuttering?" Table I shows the answer to this question.

TABLE I.

RESPONSE TO THE QUESTIONNAIRE

Questionnaire Sent To:		Replies			
	No.	Answer Yes	Answer No	Total	Percent Replying
Residential Schools	65	16	34	50	76.9
Denominational and Private	20	1	13	14	70.0
Public Day Schools	121	25	51	76	62.8
Total	206	42	98	140	67.9

These replies have furnished information about 55 deaf stutterers. Table II shows the distribution of these cases by sex according to percentage of hearing loss and age of onset of deafness.

TABLE II

DISTRIBUTION OF DEAF STUTTERERS BY SEX ACCORDING TO PERCENTAGE OF HEARING LOSS AND AGE OF ONSET DEAFNESS

Hearing Loss	Age of Onset of Deafness (by years)													
	Birth	1	2	3	4	5	6	7	8	9	10	11	14	Unknown
90-100	M-5				M-20					M-7				M-6
	M-10													M-30
	M-35													F-50
80-89	M-43				F-32			F-26						
					M-31									
70-79	F-25											M-23		
	M-28													
60-69				M-37			M-39		M-15					?-53
50-59	M-42		M-17			M-36	M-41	F-3		M-11			F-22	
	M-52							M-12						
40-49		M-29			M-14									
					M-33									
					M-47									
30-39	M-38		M-45									?-48		F-8
20-29	M-13					M-44	M-40	M-27	M-24					M-46
Partial	M-21		M-16								M-18			M-19
														M-49
														?-54
														?-55
Unknown	M-51					M-2								M-1
														M-4
														M-9
														M-34

M and F refer to "male" and "female." The numbers following M or F designate case numbers. The question marks in cases 48, 53 54 and 55 indicate that sex was not designated.

Table III displays enrollment according to sex, for the year 1937-38, in the schools replying to the questionnaire.

TABLE III.

ENROLLMENT BY SEX IN DEAF SCHOOLS REPLYING TO QUESTIONNAIRE

Schools	Boys	Girls	Total
Public Residential (10 reported no numbers)	5,381	4,529	9,910
Denominational and Private (1 reported no numbers)	467	411	878
Public Day Schools (5 reported no numbers)	1,588	1,315	2,903
Total	7,436	6,255	13,691

In presenting this new problem of the existence of stuttering among the deaf, it is interesting to observe in Table I that the teachers in 70 per cent of the institutions replying had never heard of such cases, and many offered various reasons, above mentioned, why this phenomenon would not appear.

Table III shows that among schools replying to the questionnaire, there are 1,181 more boys than girls enrolled. The American Annals of the Deaf[2] lists a total enrollment of 10,491 boys and 8,787 girls, a difference of 1,704. This sex ratio has no doubt been critically observed before. It is especially interesting in view of the sex ratio found in stuttering. West[3] says, "The ratio of stuttering in boys and girls above 36 months of age varies all the way from 3 to 1 to 8 to 1." Table II shows a similar sex difference among deaf stutterers: forty-four males, seven females, four cases in which sex is not reported; a ratio of slightly over 6 to 1.

The percentage of the 55 stuttering cases among the total deaf population cannot be accurately determined from the present figures. The principal factor tending to make such a percentage too high is the fact that the total deaf school population listed is for this year, whereas many of the stuttering cases listed are not at present in school; the principal factor tending to make such a percentage too low is the fact that the total figure includes the presence of that group of deaf children, not yet determined, who have no speech at all, and hence should not be included. The attempt is now being made to obtain a reliable percentage. The percentage of stuttering among the total hearing population is about one per cent;[1] it would be significant to compare with this the incidence of stuttering among the deaf.

Another interesting feature of this study involves causation. High fever has been considered a frequent etiological factor in stuttering. Since a large number of the acquired cases of deafness are caused by diseases which are accompanied by high fever, one might expect therefore a higher percentage of stuttering among the deaf than among the normal. Table II shows, however, only 44 cases of stuttering among individuals with acquired deafness. For this reason it seems likely that further study of these cases might be valuable.

Table II displays the distribution of deaf stutterers according to degree of hearing loss and age of onset of deafness. It is almost impossible to determine the region beyond which the degree of hearing ceases to be serviceable for the acquisition of speech. It may be reasonably assumed, however, that the six cases showing a hearing loss of 70 per cent and above from birth may be considered totally deaf for purposes of speech. While it is undesirable upon the basis of this preliminary report to generalize, nevertheless this data presents two significant facts: it establishes the existence of deaf stutterers, and it calls into question any theories about the causation of stuttering which would preclude the possibility of its occurrence among deaf children.

STATE TEACHERS COLLEGE.

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XLIX

RECENT ADVANCES IN THE DIAGNOSIS AND TREATMENT OF DEAFNESS*

GEORGE E. SHAMBAUGH, JR., M.D.

CHICAGO

The diagnosis of curable from incurable deafness depends to a large extent upon the hearing tests. Certain advances in the use and interpretation of hearing tests have a very practical application in the diagnosis of two important kinds of curable deafness. One of these, secretory otitis media, is a form of conduction deafness that has been known for a long time. The other, exudative labyrinthitis, is a form of inner ear or perceptive deafness that was first described two years ago.

For many years we were taught that in pure conduction deafness there is a greater loss of hearing for low tones than for high tones. Fig. 1 is the hearing tests of a patient with stapes fixation from otosclerosis. Notice the pronounced loss for low tones with almost normal hearing for the very high tones. Compare this with Fig. 2, which is from a patient with a secretory otitis media. Notice that the loss of hearing for the high tones is a great as for the low tones. That this was not due to any inner ear lesion is proved by the fact that evacuation of the fluid from the middle ear by paracentesis and inflation resulted in a prompt restoration of normal hearing to all tones.

Page 638 compares the hearing tests of a group of patients with secretory otitis, with those of a group of patients with otosclerosis. The diagnosis of secretory otitis was made by the observation of clear yellow serum shining through a normal drum membrane with bubbling on catheter inflation and restoration of normal hearing after paracentesis and inflation. Notice that in each case with fluid in the middle ear the loss for high tones equalled that for low tones, while in otosclerosis the hearing for high tones was much better than for low.

What is the explanation for this difference? The explanation, I believe, is that low pitched sounds reach the labyrinth through the

*Presented before the meeting of the Chicago Laryngological and Otological Society, February, 1938.

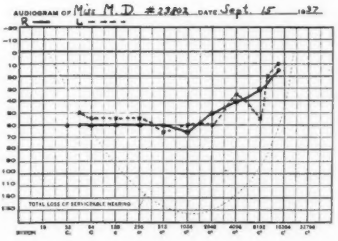


Fig. 1.

Audiogram of a case of otosclerosis. Note good hearing for high tones. (Solid line—R. ear, dotted line—L. ear.)

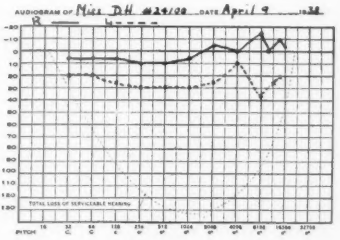


Fig. 2.

Audiogram of a case of secretory otitis media (left ear). Note loss for high as well as low tones. (Solid line —R. ear, dotted line—L. ear).

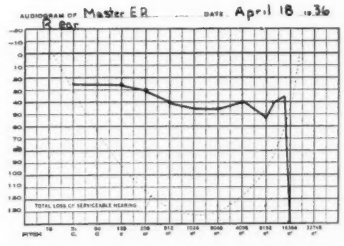
drum membrane and ossicular chain through the oval window, while high pitched sounds enter through the round window. If this is correct, then a traumatic rupture of the drum membrane should result in the same tests as otosclerosis, whereas a suppurative otitis media should give the same picture as a secretory otitis (Fig. 4).

The second type of curable deafness, namely, exudative labyrinthitis, depends for diagnosis upon the demonstration of diplacusis, which is the symptom where a given tone is heard at a different pitch in the two ears. The symptom of diplacusis has been mentioned occasionally in the otological literature for many years, but its significance for the diagnosis and treatment of a certain type of deafness was not brought out until three years ago, when I called attention to a clinical syndrome consisting of unilateral low tone nerve deafness with diplacusis and with attacks of vertigo.

According to the accepted theory of hearing, each tone stimulates its own group of hair cells in the cochlea and its own nerve fibres in the eighth nerve transmit the impulse to the brain (Fig. 5). Lesions of the conducting mechanism can modify the intensity with which sounds reach the inner ear but, of course, cannot alter the quality or pitch. On the other hand, changes in the vibrating membrane consisting of an edema, an adherent blood clot or an inflammatory exudate (Fig. 6) will result in an alteration in the vibratory response of this membrane and a change in the quality or pitch of the tone. It is significant that where diplacusis is present there is almost always a change not only in pitch but in quality, so that in

ACUTE CATARRHAL OTITIS MEDIA	OTOSCLEROSIS
M. H.—Whisper R 22, 18 in. 66, 6 in. 32 fork much shortened 64 fork slightly shortened 2048 fork scarcely heard Rinné positive	Dr. S. L. M.—Whisper L 22, 6 ft. 66, 10 ft. 32 fork not heard 64 fork much shortended 2048 fork slightly shortened Rinné negative
E. R.—Whisper R 22, 1 ft. 66, 6 in. 32 fork moderately shortened 64 fork slightly shortened 2048 fork slightly shortened Rinné equal	Mrs. W.—Whisper R 22, 1 ft. 66, 2 ft. 32 fork much shortened 64 fork moderately shortened 2048 fork normal Rinné negative
C. D.—Whisper R 22, 2 ft. 66, 4 ft. 32 fork moderately shortened 64 fork normal 2048 fork slightly shortened Rinné shortened positive	M. L. Q.—Whisper L 22, 18 in. 66, 2½ ft. 32 fork not heard 64 fork much shortened 2048 fork normal Rinné negative
F. C.—Whisper R 22, 6 in. 66, close to ear 32 fork moderately shortened 64 fork slightly shortened 2048 fork much shortened Rinné not recorded	Mr. N.—Whisper L 22, 6 in. 66, 1 ft. 32 fork not heard 64 fork scarcely heard 2048 fork slightly shortened Rinné negative
A. L. H.—Whisper R 22, 18 in. 66, 2 ft. 32 fork slightly shortened 64 fork slightly shortened 2048 fork slightly shortened Rinné equal	Mrs. B.—Whisper R 22, 1 ft. 66, 3 ft. 32 fork not heard 64 fork not heard 2048 fork slightly shortened Rinné negative
Both windows impeded: All tones affected. Only moderate impairment of low tones. Rinné: shortened positive, equal, rarely negative.	Stapes only impeded: Low tones predominantlyaffected. Marked impairment of low tones. Rinné: negative.

Hearing tests on 5 cases of acute catarrhal otitis media compared with . . . 5 cases of otosclerosis with similar degrees of hearing impairment.

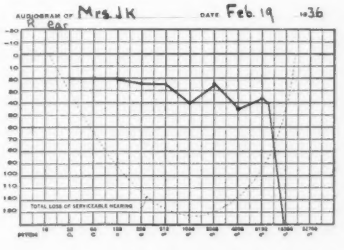


Acute suppurative otitis media right. Note loss for high as well as for low tones.

Whisper 44, 4 in.
66, close to ear

Forks: 32 moderately shortened
64 slightly shortened
2048 slightly shortened

Rinné negative, Schwabach prolonged

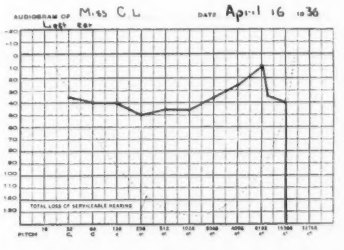


Acute catarrhal otitis media right. Note loss for high as well as low tones.

Whisper 24, 6 in.
66, 6 in.

Forks: 32 slightly shortened
64 very slightly shortened
2048 slightly shortened

Weber right Rinné positive
Schwabach prolonged

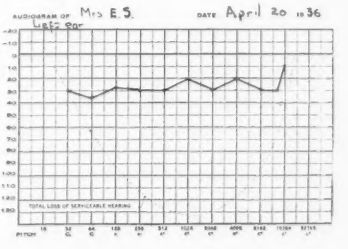


Otosclerosis (poorer hearing ear) left. Note loss for low tones, good hearing for high tones.

Whisper 24, 6 in.
66, 2 ft.

Forks: 32 not heard
64 much shortened

Rinné negative Weber left
Schwabach prolonged



Traumatic rupture of drum membrane left. Note loss for low tones with good hearing for high tones.

Whisper 24, 1 ft.
66, 2 ft.

Forks: 32 not heard
64 much shortened

Rinné negative, Schwabach prolonged

Fig. 4.

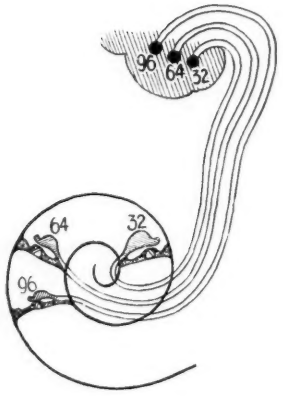


Fig. 5. Each tone stimulates a different area in the cochlea and a definite set of nerve fibers which transmit the impulse to the auditory center in the brain.

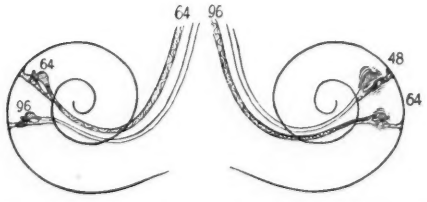


Fig. 6. In the normal ear the note of a fork of 64 double vibrations stimulates its special portion of the organ of Corti and its set of nerve fibers. In the affected ear the vibrating membrane has been altered so that the area which previously responded to the fork of 96 double vibrations now responds to that of 64 double vibrations, the tone of the latter fork being heard in the brain as one of 96 double vibrations, resulting in diplacusis.

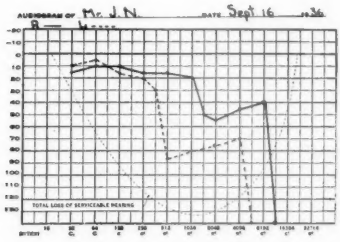


Fig. 7. Audiogram of probable hemorrhage in basal coil of left cochlea. (Solid line—R. ear, dotted line—L. ear).

Left
32 fork: slight shortened
64 fork: normal
2048 fork: scarcely heard
Diplacusis: 64, 108, 128, 2048; all slightly higher in left.

the affected ear the tuning forks have a rough, impure, harsh sound as compared to the smooth, musical tone in the normal ear.

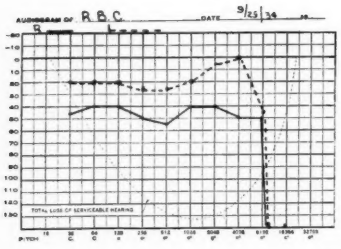
I have had one case of diplacusis which apparently was due to a small hemorrhage into the labyrinth.

REPORT OF CASES

CASE 1.—Mr. J. N. (Fig. 7), aged 70, awoke one morning deaf in the left ear with ringing in this ear and with nausea, vomiting and ataxia. Previous to this his left ear had been his better hearing ear. The hearing in the left ear improved somewhat during the next two months, and since then has remained stationary. Hearing tests reveal a sharply localized defect in the left ear with diplacusis for the low pitched forks which are heard at a higher pitch in the left ear. Because of his age, the single attack of vertigo and the sharply localized defect involving chiefly the high tones, with diplacusis for the low tones, this was probably a small hemorrhage into the basal coil of the cochlea, destroying most of the hearing in this region and sparing the apical coil. Enough fibrin or red blood cells became deposited on the vibrating membrane of the apical coil to weight it down and cause a diplacusis.

Nearly all of the remaining cases of diplacusis that I have seen have been associated with an inner ear deafness which fluctuated from time to time, and usually with repeated attacks of vertigo.

CASE 2.—Mr. R. B. C. (Fig. 8), aged 56, was brought to my office on May 24, 1934, supported between two men, too dizzy to stand. His trouble had begun abruptly three months before with an attack of dizziness, nausea and vomiting, with ringing and defective hearing in the right ear. The dizziness lasted only a



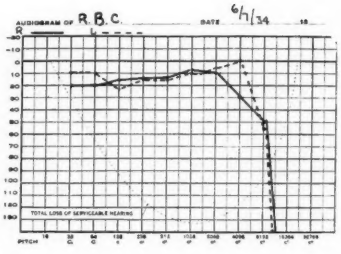


Fig. 8.

Audiogram before tonsillectomy. Low tone nerve deafness in right ear.

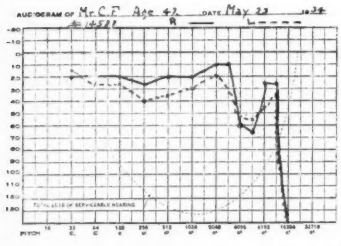
	R	L
Whisper	close	2 M
32	scarcely heard	Normal
64	slightly shortened	Normal
2048	slightly shortened	Normal

Rinné positive right and left
Weber to left
Diplacusis: 64, 108, 128, 216, heard higher in right ear

Audiogram one week after tonsillectomy.

	R	L
Whisper	3½ M	4 M
32	Normal	Normal
64	Normal	Normal
2048	Normal	Normal

Rinné positive R and L. Weber to left
Diplacusis still present, none six months later



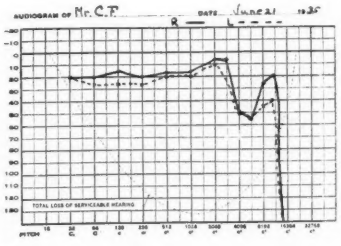


Fig. 9.

Audiogram before tonsillectomy. Case of low tone nerve deafness in left ear.

	Left
32 fork	much shortened
64 fork	slightly shortened
2048 fork	slightly shortened

Rinné positive left
Schwabach shortened
Diplacusis: 64, 108, 128. Patient not musical; cannot say whether higher or lower.

Audiogram four weeks after tonsillectomy.

	Left
32 fork	Normal
64 fork	Normal
2048 fork	Normal

Diplacusis for 64, 108. 128

day, but the tinnitus and partial deafness had persisted for three months. Three days before coming to my office he experienced his second attack of dizziness, and on the morning of his visit the third attack occurred.

At the time of examination he was still dizzy and nauseated with a spontaneous nystagmus toward the left (good) ear. The drum membranes were normal and the eustachian tubes patent. Hearing tests showed a pronounced defect in the right ear where whisper was heard at the ear. The tuning fork tests showed clearly an inner ear type of defect, but with greater impairment for the low tones than for the high. This patient, therefore, had that unusual condition, a low tone nerve deafness. On testing for diplacusis the low forks were heard at a higher pitch in the right ear.

Since there had been three separate attacks of vertigo, an inflammatory lesion in the cochlea was diagnosed. The fact that the forks were heard higher pitched in the diseased ear was consistent with the assumption of an inflammatory exudate adhering to the vibrating membrane, causing an area which previously responded to higher tones now to vibrate more slowly and to respond to a lower fork.

On this assumption a search for a focus of infection revealed imbedded and reddened tonsils, from one of which a drop of pus was expressed. One week after tonsillectomy the tinnitus and deafness, constantly present for three months, had entirely cleared up and there have been no further attacks of vertigo.

CASE 3.—Mr. C. F. (Fig. 9), aged 42, complained of roaring and defective hearing in the left ear for one year. Hearing tests showed a bilateral nerve deafness with a greater loss for the low forks than for the high in the left ear, and with diplacusis, the tones being of a different quality in the two ears, though the patient cannot distinguish pitch. Four weeks after tonsillectomy the hearing had returned almost to normal and the roaring which had been present for a year was gone.

CASE 4.—Mr. W. R. W. (Fig. 10), aged 42, complained of dizzy attacks for five years and of roaring and defective hearing in the left ear for two weeks. An orchestra had sounded distinctly out of tune. Hearing tests showed a low tone nerve deafness in the left ear with diplacusis. Tonsillectomy was followed by normal hearing six weeks later and the dizzy attacks have never recurred.

CASE 5.—Mr. W. B. S. (Fig. 11), aged 56, complained of vertigo for six days. He was not aware of a hearing defect, but tests showed a low tone nerve deafness in the right ear with diplacusis. A bladder infection improved and three weeks later the hearing was normal and there has been no more vertigo.

CASE 6.—Miss U. (Fig. 12), aged 40, complained of a high pitched tinnitus in the left ear for seven weeks. The hearing was normal and I was unable to explain the tinnitus. There were two dead teeth, but the x-ray showed no abscess, so these were not disturbed. Three months later the patient returned, because for two weeks the tinnitus had been much worse, with a distinctly jarring discomfort in this ear on exposure to loud sounds. Hearing tests disclosed a low tone nerve deafness with diplacusis for the low forks. The diagnosis of exudative labyrinthitis was made and removal of the dead teeth advised. This was done, one tooth being found abscessed. Within a week the ear symptoms, present for five months, began to improve and soon disappeared. Three months later the patient was tired

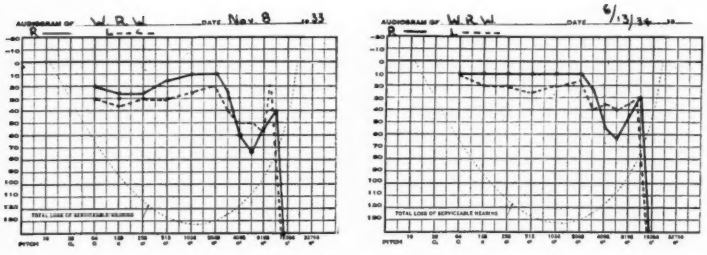


Fig. 10.

Audiogram of case of low tone nerve deafness.

Left
32 fork not heard
64 fork mod. shortened
2048 fork slightly shortened
Weber right Rinné positive
Schwabach normal
Diplacusis for all tones; poor quality and lower pitch in left ear.

Audiogram six weeks after tonsillectomy.

Left
32 fork normal
64 fork normal
2048 fork slightly shortened
No diplacusis

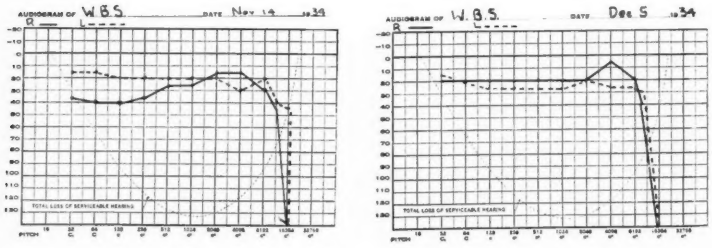


Fig. 11.

Audiogram of case of low tone nerve deafness.

Right
Nov. 14: 32 fork scarcely heard
64 fork sl. shortened
2048 fork normal
Rinné positive, Schwabach shortened
Diplacusis: 32, 64 higher in right

Audiogram three weeks later.

Right
Feb. 2: 32 fork normal
64 fork normal
No displacusis

A—Normal hearing at first examination

Fork:	R	L
32	very sl. shtd	slightly shortened
64	normal	normal
2048	normal	normal

B—Low tone nerve deafness with diplacusis, left ear

Fork:	R	L
32	very sl. shtd	scarcely heard
64	normal	mod. shortened
2048	normal	normal

C—Normal hearing, diplacusis largely disappeared

	R	L
32 fork	normal	normal
64 fork	normal	normal
2048 fork	normal	normal

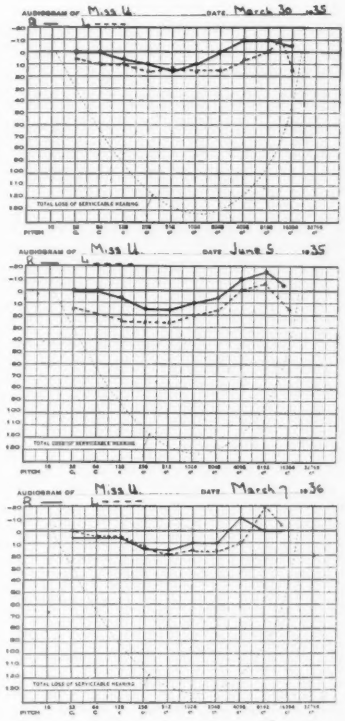


Fig. 12.

out and the ear symptoms recurred for several weeks, with three separate attacks of vertigo. Since then she has been free from trouble.

Case 7.—Mrs. P. W. P. (Fig. 13), had severe tinnitus in the left ear with a low tone nerve deafness and diplacusis. Removal of an infected tonsil tag and of infected teeth resulted in a temporary improvement, then a relapse. She was advised to have infected lateral pharyngeal bands removed, but delayed until one day she had a typical Ménière's attack. The lateral bands were removed soon after, with complete recovery from all ear symptoms. A few months ago she returned because of a slight recurrence of tinnitus, deafness and diplacusis following a mild sore throat. This cleared up in a few weeks.

These cases of low tone nerve deafness with diplacusis and vertigo have certain characteristics. The age incidence, the usually unilateral involvement, the tendency to recurrences, the tendency of the hearing to fluctuate and, finally, the apparent relation to focal

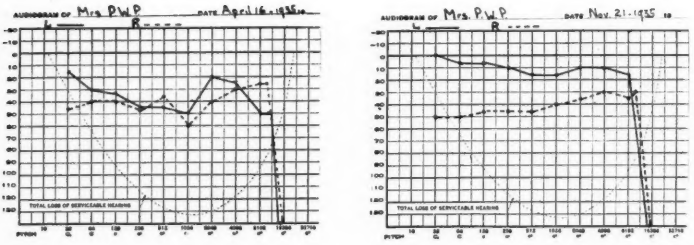


Fig. 13.

A—Low tone nerve deafness in left ear with diplacusis. Conduction deafness in right ear.

Left
Whisper 22, 3 ft.; 66, 4 ft.
32 moderately shortened
64 slightly shortened
2048 normal
Schwabach normal
Rinné strongly positive
Diplacusis: 64, 108, 128 higher in L.

B—Normal hearing in left ear. No diplacusis.

Left
Whisper 22, 12 ft.; 66, 12 ft.
32 normal
64 normal
2048 normal
Schwabach prolonged, Rinné pos.
No diplacusis

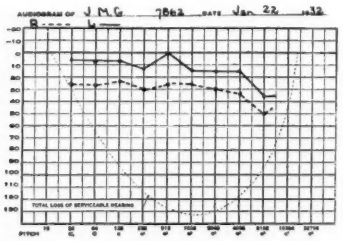
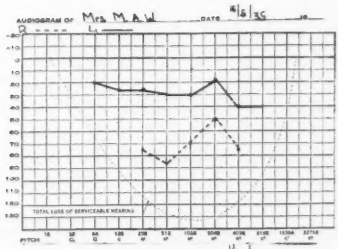


Fig. 14. Audiogram of case of serous labyrinthitis left ear. Solid line—R. ear, dotted line—L. ear.

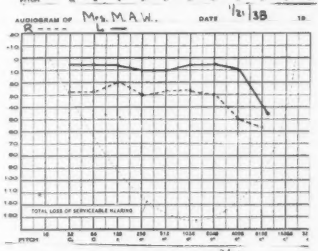
	R	L
Whisper	9 feet	1 foot
C_1	normal	not heard
C	normal	much shortened
c^4	normal	slightly shortened

Diplacusis for C, A, which are heard several tones higher in left ear and with a definitely rough, non-musical quality.

Weber left
Schwabach R shortened
L normal
Rinné pos. R and L



Whisper R 24, 2 ft. L normal
66, 1 ft.
C_1 R not heard L sl. shtd.
C R much shtd. L sl. shtd.
c^1 R normal L normal
Weber A left c left
Schwabach definitely shortened
Rinné positive R and L
Diplacusis: C, c, A, a^1 all heard higher pitched in R, several notes higher for low forks, less for a^1



Whisper R 24, 4 M L normal
66, 1 M
C_1 R much shtd. L normal
C R mod. shtd. L normal
c^1 R normal L normal
Weber R ? Schwabach shortened
Diplacusis as before but pt. thinks it is less pronounced.

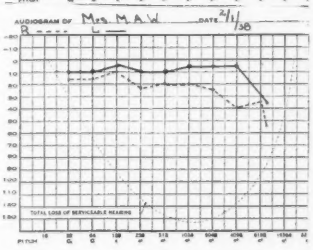


Fig. 15. Low tone nerve deafness (left ear) with diplacasis and vertigo, showing marked improvement in hearing after removal of a focus of infection in the maxillary sinus.

infection, all resemble very closely iritis. I suspect that the pathology is similar to iritis with cells in the fluid, with a tendency for early and mild cases to recover completely, whereas older and more severe cases result in a permanent impairment of function. Since none of my patients have died, the exact pathology must remain for the present hypothetical. However, if my assumption of the pathology is correct, then we should expect that a serous labyrinthitis secondary to an acute suppurative otitis media with cells and inflammatory exudate in the labyrinth would result in a diplacusis. For a long time I searched for a case of serous labyrinthitis to try out the tests for diplacusis, and I recently found a case.

CASE 8.—Miss J. M. G. (Fig. 14), aged 57, had a severe acute otitis media in the left ear nine months ago, resulting in an apparently total deafness in that ear and pronounced ataxia lasting three weeks. The hearing in this ear has returned somewhat since then, but is still defective. She noticed that an orchestra seemed badly out of tune soon after the onset of her trouble, and this symptom, while still present, is improving. At the present time she has a large, dry, central perforation in the left ear. Hearing tests show a diplacusis, tones being heard with a definitely rough, non-musical quality and at a different pitch as compared to the normal ear.

CASE 9.—Three weeks ago Mrs. M. A. W. (Fig. 15), came to me because of nasal symptoms which proved to be due to a chronic purulent maxillary sinus infection. She gave the additional history of defective hearing and roaring in the right ear for six or seven years. Four years ago she began to have severe attacks of vertigo with nausea, vomiting and ataxia. She visited a University clinic, where an audiogram was obtained (Fig. 15—4-5-35). The tuning fork tests showed that this was a nerve deafness, but she was not tested for diplacusis. She was put on Furstenberg's low salt diet and had no more attacks of vertigo. When I tested her hearing three weeks ago I found a low tone nerve deafness with diplacusis. The interesting thing is that clearing up this chronic maxillary sinus infection by a few irrigations through the natural ostium resulted in this striking improvement in hearing, so that now she hears better in this ear than for at least six years.

CONCLUSIONS

Diplacusis is a not infrequent finding if it is specifically tested for. When it is found associated with low tone nerve deafness and vertigo, particularly where the hearing fluctuates from time to time, the patient probably has an inflammatory lesion in the membranous labyrinth which we may call exudative labyrinthitis and which is comparable in its etiology, pathology and clinical course to iritis.

122 SOUTH MICHIGAN AVENUE.

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CHORDOMA: WITH REPORT OF TWO CASES*

ROBERT F. RIDPATH, M.D.

PHILADELPHIA

Chordoma is a rare and usually fatal tumor which arises from the fetal notochord. This structure is a cord of cells around which the vertebral column develops, regularly persisting in infants in the centers of the intervertebral disks. Portions of chorda tissue commonly continue to be present at the base of the skull and in the coccyx; this tissue gives origin to certain small tumors which Virchow described as "ecchordosis spheno-occipitalis."

As the cells multiply they become vacuolated and contain a mucin-like substance. The walls of these cells break and a syncytium forms in a gelatinous matrix. As the vertebræ develop, the notochord becomes segmented. The intravertebral portion remains as the nucleus pulposus of the intervertebral disks. In spite of the fact that notochordal remnants are present in the nucleus pulposus of all intervertebral disks throughout life, very few chordomas have been found between the base of the skull and the sacrum.

Small masses of soft, jelly-like tissue arising from the clivus blumenbachii (the sloping surface between the sella and foramen magnum composed of the sphenoid and occipital bones) were described by Luschka in 1856. Virchow, elaborating upon the subject, attributed these to degeneration of the cartilage and gave them the name "ecchondrosis physaliphora." H. Muller was the first to ascribe their origin to notochordal tissue. He showed that the notochord extended to the spheno-occipital synchondrosis and that "nests" of tissue could be demonstrated in the basilar cartilage. These were similar in structure to the nucleus pulposus of the intervertebral disks. In 1894, Ribbert gave the tumor the name chordoma. He found chorda remnants in the bony tissue beneath the dura mater of the clivus and held that they produced tumors only when misplaced and lying outside the bony encasement.

Recently, several excellent reviews have served to direct attention to this interesting group of tumors. A chordoma is a neoplasm

*Presented before the American Laryngological Association, Atlantic City, May 3, 1938.

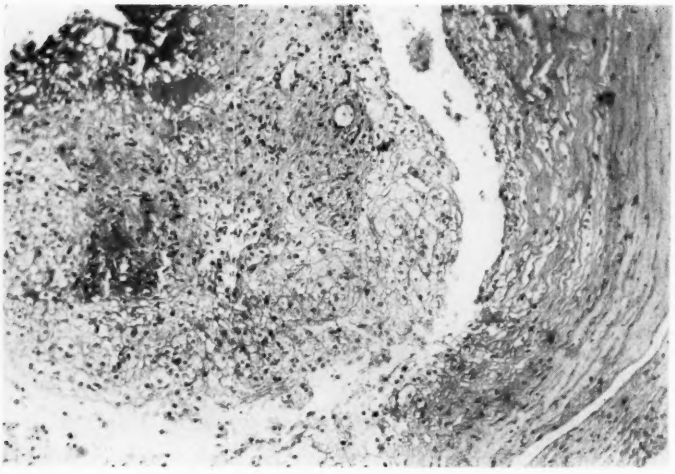

Fig. 1. Spheno-occipital chordoma, low power, 100 diameters. Note just to the right of the center, large vacuolated cells resembling cartilage cells. They differ from cartilage in the absence of inter-cellular substance.

of notochordal origin. It arises from the embryonic remains of the chorda dorsalis, the primitive axial skeleton of a vertebrate, which is derived from the entoderm. Such cells may escape encasement by cartilage during development or conceivably as a result of trauma. Theoretically a chordoma may be produced at any point along the axial skeleton where notochordal cells are found.

The chordomas have been classified as (1) cranial, (2) vertebral, and (3) sacro-coccygeal. They have been reported as occurring in various locations in the following order of frequency: in the sacro-coccygeal region, in ninety-one cases; in the spheno-occipital region, in fifty-two cases; in the cervical portion of the spine, in twelve cases; and in other locations rarely.

Of the fifty-two reported cases of spheno-occipital chordoma, ten of those being naso-pharyngeal, only twelve have been reported in the American literature. However, that is probably due to the laxity on the part of us Americans, as I know of five in Philadelphia which have not been reported. In each of the locations mentioned the tumor presents a fairly constant symptomatology.

As we are considering these tumors primarily from the nose and throat standpoint, we will go a little more into the detail of that aspect, taking up first the embryology.

The developing notochord extends as far forward as the buccopharyngeal membrane, and temporarily remains attached to its caudal wall as the hypophyseal pocket forms. The chorda makes a single large sigmoid curve in the midsagittal plane of the basilar plate of the skull, lying near the dorsal surface of the cartilage at three points: in the hypophyseal fossa, a short distance caudal to the fossa and anterior to the foramen magnum. It is near the ventral surface midway between the hypophyseal fossa and the foramen magnum. As the base of the skull is molded, the posterior end of the cranial part of the chorda is forced backward and dorsal, so that it lies on the occipital plate in a dorsal groove anterior to the foramen magnum. The anterior extremity is forced forward so as to lie between the cartilage and perichondrium of the dorsum sellæ. The middle part comes to lie between the cartilaginous occipital plate and the dorsal wall of the pharynx. This middle part often presents a number of kinks, branches and irregularities. Frequently it is embedded in the retropharyngeal tissues, and its cells may intermingle with the epithelial cells of the pharynx.

As far as incidence of these tumors is concerned, the majority caused the first symptoms in the third, fourth and fifth decades of life. The average age was 36 years. The youngest patient was one day, and the oldest 82 years. The tumor was found more frequently in males than in females, in the ratio of three to two. The factors which excited neoplastic proliferation were unknown, but the frequency of the history of trauma deserved emphasis.

The differentiation of benign chordomas from malignant chordomas as a rule, is not difficult. In typical instances the benign tumors are attached to a small median exostosis of the clivus by a narrow stalk which perforates the dura. The stalk is not always found, and in these instances the tumor lies in the subdural space or nestles beneath the dura against the bone of the clivus. These benign tumors are relatively common, being present in 2 per cent of autopsies, according to European authors. The malignant tumors vary in size from small (2 to 3 cm.) to massive neoplasms, the largest of which was 11 x 7 x 6 cm. In form they are pedunculate, mushroom-like, globular or diffuse. On the clivus they are generally covered by dura and in the nasopharynx are limited by the mucosa. The mucous membrane of the nasal fossa, paranasal sinuses and

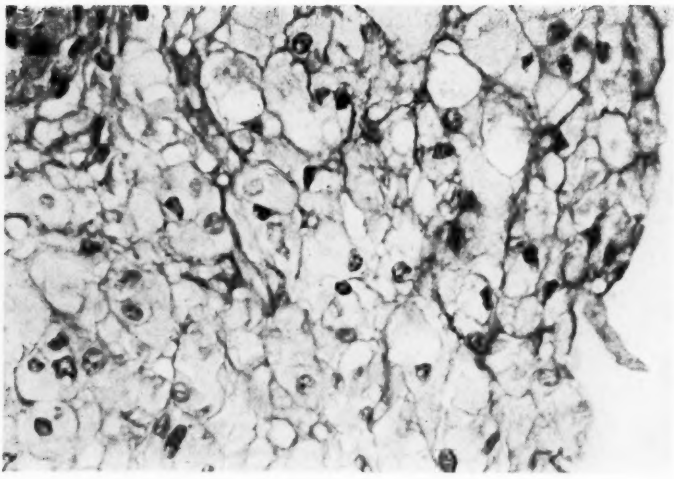

Fig. 2. Spheno-occipital chordoma, magnified 450 diameters. Area referred to in Fig. 1. Note large vacuolated cells containing mucinous material.

pharynx is seen elevated in a characteristic bulging manner by the slowly infiltrative, expanding growth.

The majority of cranial chorda masses arise from the clivus in the region of the spheno-occipital synchondrosis. The typical ventral extension is in the direction of the nasopharynx. It is common for the basilar plate to be infiltrated and partially destroyed. Somewhat less frequently a smooth, round mound of tumor elevates the mucosa in the midline of the posterior epipharyngeal wall. These growths extend for variable distances anteriorly and caudally, although as a rule they are restricted to the vault of the epipharynx. In exceptional instances tumor tissue pressed through the posterior choana into the nasal fossæ and even into the maxillary antra. Contrary to expectations, the volume or extent of the nasopharyngeal growths does not parallel the size of the intracranial neoplasm. The regional petrous portions of the temporal bones, the ethmoid sinuses as far forward as the crista galli, and the osseous walls of the orbital cavities occasionally are involved. It is fairly characteristic of this widespread anterior growth to be associated by ventral invasion into the walls of the nasal fossa and into the superior maxillary bones,

with the tumor encroaching on the nasal cavities, the maxillary antra and the soft tissues of the face.

The histologic picture is characteristic, but chordoma must be differentiated from two common types of tumor, myxochondroma and colloid carcinoma. Pathologically variable pictures are presented in the different phases of chordoma. The early structure resembles hyaline cartilage, but without intercellular substance. The cells are large, rounded, vacuolated and hyperchromatic, and may even resemble epithelium. An alveolar structure is seen in more active cases. Much mucinous material may distend the cells and infiltrate the invaded tissues. Syncytial masses of vacuolated cytoplasm may be prominent.

The symptomatology from the rhinolaryngological viewpoint is fairly constant. The nasopharyngeal symptoms, although they accompany or follow the onset of headaches and visual disturbances, may be the first evidence of the disease. As a result of the obstruction of the nasopharyngeal passages by the tumor, difficulty in breathing, nasal voice and mucoid discharge from the nose develop, this with nasal bleeding. Involvement of the sinuses and loss of weight are constant factors.

In the differential diagnosis we rely almost solely on the aid of roentgen examination and biopsy. However, in certain cases, a proper analysis of the signs and symptoms should lead to a surprising number of accurate diagnoses. In recapitulation, the salient clinical features may be summarized as follows: A gradual onset of nasopharyngeal and neurologic symptoms in middle-aged patients; the presence of an obstructing, midline, nasopharyngeal tumor; progressive bilateral palsies of the cranial nerves, which may be quite widespread with little or no evidence of intracranial hypertension; sensory as well as motor manifestations, and symptoms arising from compression of the brain stem or the upper cervical portion of the spinal cord.

It would seem wise to enumerate the common mistakes and to dwell briefly on a few of the more important points of differentiated diagnosis. There are many good reasons for confusing a chordoma with a craniopharyngioma (tumor of Rathke's cleft and hypophyseal stalk). The manifestations referable to the cranial nerves, especially the defects in the visual fields and the roentgenographic changes may be identical. There may even be supracellar calcification, such as is common in a craniopharyngioma. The adenomas and carcinomas of the hypophysis may destroy a considerable amount of bone, and in order to complete the imitiation of a chordoma they may grow

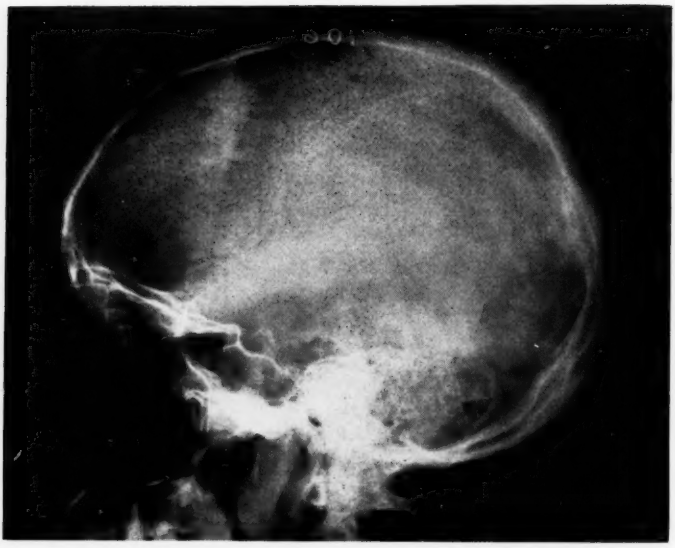

Fig. 3. "There is a very large tumor in the nasopharynx. This is continuous with a soft tissue mass which fills about 80 per cent of the sphenoidal sinuses. There is a definite defect in the basilar process of the occipital bone in the neighborhood of the clivus." *(X-ray report of Dr. L. J.)*

into the nasopharynx. Therefore, one distinct point of difference between the anterior group of chordomas and the hypophyseal tumors deserves emphasis. In the former there is little or no evidence of hypophyseal dysfunction. As craniopharyngiomas usually occur in children, chordomas are less frequently confused with these tumors. Parasellar meningiomas, although they rarely extend into the nasopharynx, should be considered in the differential diagnosis.

Chordomas which involve the nasopharyngeal passages almost invariably are overlooked or are considered as one of the more common nasopharyngeal tumors. The rare juvenile nasopharyngeal fibroma may arise in the same situations and may grow in much the same manner as a chordoma. It may extend into the cranial cavity and cause various neurologic symptoms. However, these tumors develop before the twenty-fifth year. There is a tendency for the tumor to disappear spontaneously, chiefly toward the end of the second decade of life. Atypical chondromas, chondrosarcomas and

myxosarcomas in this region may offer diagnostic difficulties not only to the clinician but also to the roentgenologist and pathologist. Other local tumors such as pharyngeal dermoid cysts and teratomas usually occur in infants and children. Although a primary carcinoma of the nasopharynx may invade the base of the skull and involve the cranial nerves, metastases are almost invariably found in the cervical lymph glands.

As regards prognosis and treatment, little offer of hope can be promised. Chordomas invariably prove fatal in all instances in which the patients are under observation for a long period. The prognosis of so-called nasopharyngeal tumors is variable, depending largely on the presence or absence of cranial involvement, or involvement of the spinal cord. One patient, in the literature, had a history of difficult breathing through his nose for seven years. A tumor projecting from the posterior wall of the nasopharynx was removed. It recurred a few months later. A second operation was performed four and a half years after the first. Five months later the patient was well. In a similar instance the patient returned with a recurrence four and a half years later.

It seems that operation, as a rule, offers only temporary amelioration of symptoms. Yet it is possible that pendunculate, encapsulated, noninvasive tumors may be completely eradicated by excision. Supplementary roentgen and radium treatment are of no established benefit.

REPORT OF CASES

CASE 1.—Dr. L. J., age 38, white, a Haitian, who entered the Temple University Hospital November 16, 1936. His chief complaints were: Occipital headaches for the past three years. Nasopharyngeal catarrh with mucopurulent and bloody discharge and more or less nasal respiratory insufficiency. There was some dyspnea at times. Coryza two months ago. He had lost twelve pounds in the past two months. The family history is negative. The past medical history reveals that under local anesthesia on September 16, 1936, he was operated upon for fibrous adenoids. There is also a history of a "flying piece of iron," causing a considerable bruise and cut in the scalp some years previously. The present medical history dates about three years ago when he noticed some impairment of hearing on the right side and consulted a physician, who found nothing. Three months ago severe headaches radiating to the back of the head over the occipital region became very pronounced. There also was a bloody postnasal discharge. The headaches were of such severity he was unable to eat or sleep and at this time became cognizant of beginning loss of weight.

He returned to his physician, who found by posterior rhinoscopy a large hard tumor which did not bleed on palpation. He entered the hospital and this growth was removed, and he returned to his practice after a period of four days.

Fig. 4. "The sella turcica has been practically destroyed. The body of the sphenoid bone and the entire basilar process of the occipital bone have been completely destroyed. Ventrally the destruction involves the ethmoid mass. The floors of the middle fossæ are destroyed mesially. There is absolutely no suggestion of a sphenoid sinus." *(X-ray report of Mrs. A. B.)*

Three weeks later the bloody discharge and frequent epistaxis returned and examination revealed a hard tumor mass in the same region with considerable ulceration in the upper portion. No enlargement of the glands was found. On September 16th another operation, under local anesthesia, was performed and for fifteen days the patient was free from symptoms.

However, after this short period the severe headaches and bloody discharge were more severe than before. In consultation with his physician he decided to consult Dr. Jackson at the Temple University Hospital. It is remarkable that up to this time no x-ray or laboratory studies were made. Dr. Jackson kindly referred the patient to me and my examination on November 18, 1936, is as follows:

"There is quite a large tumefied mass in the meso and epipharyngeal space, more on the right side and toward the midline than on the left. Postrhinoscopic view shows that this mass extends to the tip of the choana and is associated with the base of the sphenoidal sinus, it is hard and immobile. Nasal examination shows an infection of the sphenoidal cavity with some bloody, tenacious pus coming from the ostium. The mucous membrane in this area is decidedly inflamed and granular. The particular kind of growth I am unable to designate, but am inclined to think of chordoma, as it appears to start from the base of the sphenoidal sinus or at least

from near this area and probably extends downward and upward into the sphenoidal cavity." A biopsy taken at this time revealed a typical picture of nasopharyngeal chordoma.

Dr. Chamberlain's x-ray report was as follows: "There is a very large tumor in the nasopharynx, as seen clinically, and this appears to be continuous with a soft tissue mass which fills about 80 per cent of the sphenoidal sinuses, leaving only a little air in the ventral and cephalic corner of the sphenoid sinuses. The other sinuses present a normal x-ray appearance. There is a definite defect in the basilar process of the occipital bone in the neighborhood of the clivus.

The blood examination showed: red blood cells, 4,000,000; white blood cells, 11,400; hemoglobin, 76 per cent.

The urine examination was negative with the exception of a slight trace of albumin.

The Wassermann was negative.

CASE 2.—Mrs. A. B., white, 42 years old, was admitted to the Temple University Hospital on December 30, 1934. Her chief complaints were headache and loss of vision. The family history is negative. Post medical history is irrelevant except for the fact that twenty years ago in an accident she was caught under a pile of dirt and rocks. At that time she began to have headaches which were sudden in onset and were localized over the vertex. These frequently occurred twice a day.

The present medical history begins about one year ago when she began to have constant and severe headaches, along with failing vision in the left eye. This progressed slowly, and about six months ago vision in the right eye began to be impaired. For the past few days she has been totally blind. The pain in the head begins at the base of the skull in the occipital region and radiates in a fan-shape upwards over the skull.

There has been no tinnitus at any time, but vertigo and dizziness have always been present. Incontinence of urine, mental deterioration, as well as gastro-intestinal disturbances, have been constant factors. The mental deterioration is quite obvious in attempting to take the history as she has forgotten many details regarding her past and present history which were obtained from her sister. There is a complete loss of vision. Sensation of the face is normal. There is a definite right facial weakness of the peripheral type. The jaw and tongue deviate to the left; there is no atrophy or tremor. Hearing is impaired. The voice is masculine. The hands are slightly enlarged; the feet are slightly enlarged. Heart sounds are rather faint. Reflexes of the upper extremities are equal, 3. Reflexes of the upper patella are equal, 2. Achilles reflex left, 2; right, 1. There is a suggestive Babinski on the right but not on the left. Heel to knee test and finger to nose test are well done. Muscle strength is normal. Patient is unable to sit up with arms folded across chest.

The patient is a very large obese female, about 42 years of age. The hair is scant over forehead, but normal in texture. The skin of the face is heavy, thick and wrinkled, especially around the forehead. There is proptosis of both eyes, with thick discoloration of the skin around the eyes. The pupils are not equal; the right is about two millimeters larger than the left. Corneal reflexes are acute in both eyes—not as much so in the right. Palpebral fissure on the right is larger than on the left. On moving the eyes to the right there is a definite nystagmus;

on looking to the left the nystagmus is not so great, but the external ocular movements of the left eye are poor. Upper rotation is not possible.

The roentgenographic report is as follows:

"The sella turcica has been practically destroyed. What appear to be intact anterior clinoids are found to be approximately twice as far apart as in the normal. The two middle fossae exhibit thin floors which are not divided from each other by any suggestion of a pituitary fossa, and likewise the body of the sphenoid bone and the entire basilar process of the occipital bone have been completely destroyed. The foramen magnum has lost its ventral rim completely. The mesial ends of the petrous portions of the temporal bones have been destroyed and bone has been destroyed down to the very margin of the jugular foramen on each side, but especially on the left. Ventrally the destruction involves the ethmoid mass and has completely destroyed the bone through which the optic foramina normally pass. The floors of the middle fossae are destroyed mesially and a soft tissue mass in the nasopharynx, in the position ordinarily assumed by adenoid tissuse, appears to connect directly with the tumor or at least with the cranial contents. There is absolutely no suggestion of a sphenoid sinus."

Clinical examination postrhinoscopically reveals a large soft tissue mass in the epipharynx, practically occluding this whole area. By the use of the pharyngoscope this mass is seen filling the entire choana and extending into and involving the sphenoid region.

Histological report from autopsy specimen: "Malignant Chordoma."

It is a significant fact that in no case of spheno-occipital chordoma reported has there ever been metastasis.

The patient died January 3, 1935.

The temperature varied from 97.2 to 99.3 degrees. The pulse was more or less steady from 90 to 100. Respirations, 20.

Blood examination made December 31, 1934, showed: Red blood cells, 4,320,000; white blood cells, 6,000. Color index, 9. Type II. The Wassermann was negative.

18TH AND CHESTNUT STREETS.

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LI

RHINOPHONIA MACROSTAPHYLA*

CHARLES H. VOELKER, M.A.

HANOVER, NEW HAMPSHIRE

There are two types of organic rhinophonia aperta. There is the so-called short-palate speech of rhinophonia microstaphyla, where the velum is underdeveloped,[1] and a second type, rhinophonia macrostaphyla, which will be discussed in this paper.

The larynx has been so closely associated with speech that other important functions are given very slight attention. Jackson and Jackson[2] list for the larynx nine physiological functions: respiratory, circulatory, fixative, protective, deglutitory, tussive, expectorative, phonatory and emotional. The process of evolution has eliminated the tenth function, olfactory, in man, to some extent. There is still a distention of the laryngeal valve in sniffing. But in lower animals, a large epiglottis meets the soft palate and closes off the mouth so that air is compelled to pass in and out through the nose and prohibited from entering the oral cavity. In the case of grazing animals, the animal can feed and at the same time utilize olfaction for the discrimination between suitable and unsuitable food, and the detection of enemies. In carnivora, the animal can maintain a jaw grip in bloody combat and at the same time through the nose maintain respiration and olfactory guard. In man, upright posture, together with the increase in range of exteroceptive vision and intelligence, has lessened the necessity for olfaction. Negus[3] has pointed out that the epiglottis in man has degenerated into a relatively useless part of the larynx.

Speech developed later than this evolutionary change. Russell[4] has described the phonetic functions of the epiglottis in man. It takes characteristic positions for various vowels. The cushion of the epiglottis has considerable physiological function in timbre. It, in combination with the superior laryngeal sphincter, acts to produce tight, pinched or twangy tones. Furthermore, the epiglottis acts in the production of certain pharyngeal consonants along with the glossal articulation.

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*From the Dartmouth College Speech Clinic.

The velum, on the other hand, is employed to differentiate between oral and nasal consonants. It also functions to differentiate the oral and nasal vowels. Eijkman[5] has pointed out its physiology in voice quality. If the oro-naso-pharyngeal sphincter is closed, the voice timbre is stopped up, as in adenoid voices. If the velar sphincter is distended, the voice quality is characteristic of the cleft-palate cases (uraniscophonia) or short-palate cases (rhinophonia microstaphyla). In normal voice with nasal resonance, the velar sphincter is relaxed to an opening of about four millimeters in diameter.

In the lower animals, the coaptation of the epiglottis and the velum results in definite characteristics in the timbre of their vocalizations. There are typical nasal qualities to many noisy animals. In changing from nasal to oral timbre, the animal may take what appears to be a regurgitory pose, which, however, is more correctly merely similar to that of direct laryngoscopy, and the velar sphincter contracts. The animal voice may be of three types: (1) nasal, (2) oral and nasal, and (3) oral. If the voice quality is shrill or twangy, the nasal component may sound prominent. Grunting is nasal. Squealing and snarling is a combination of oral twang and nasality. Growling may be either nasal alone or in combination with twang and partially oral. The whinny is nasal. The bellow and roar may start nasal and become oral. Yelping and howling are generally oral. Chattering is generally nasal. The olfactory function with the concomitant velar-epiglottal coaptation results in a proclivity toward nasality in the lower animals. However, the barking of microsmatic animals, such as the sea lion, where, like man, the epiglottis has degenerated, is oral.

This tenth or olfactory function of the larynx is sometimes related to rhinophonia aperta in man. A long, extended velum is frequently seen which disappears below the tongue, so that the uvula is out of sight. The close relation of the nasal passage and the larynx in this case is not a lack of degeneration of the epiglottis, but a hypertrophy of the velum. Negus has shown that the velum is also degenerate in man.

When the soft palate extends down into close relation to the larynx, the voice quality may be nasal. If there is approximate articulation of the velum and epiglottis, the laryngologist would surgically treat the anomaly. However, in the cases thus far seen, there has been no actual articulation of the epiglottis and velum, but the soft palate extended so far downward, not only medially, as in the case of hypertrophy of the uvula, but also laterally, that the voice tended to be nasal. Thus the laryngological diagnosis has been nega-

tive where the phoniatric diagnosis (using different limits) has been organic. Thus we have a voice disorder due to partial coaptation of the soft palate and epiglottis developed in hypertrophied velum patients, which is a sort of rhinophonia aperta called rhinophonia macrostaphyla.

Phonation and emotional speech are phylogenetically late, but oral language is quite recent and consists of gestures by the mouth. Distance perception is brought about as a result of air blowing past these articulatory gestures. If the air passes off through the nose rather than the mouth which is gesturing, language is impossible (except for labiomancy) and the patient is restricted to only emotional vocalization.

Rhinophonia macrostaphyla is an organic defect of voice characterized by nasal quality. The nasal voice results from the air passing off through the nasal cavity rather than through the buccal cavity. In the less severe cases, the defect is only demonstrated in certain articulations. These articulations are distorted phonetically by progressive and regressive assimilation of the nasal phonetic element's nasal quality. Thus any word with the letters *m*, *n*, or *ng* will be nasal. This is not confined to single words but is broadened to the breath group. When the symptoms are nothing more than articulatory, the defect is called rhinolalia macrostaphyla. In the case of the articulatory defect, the long velum is too awkward to make the rapid changes characteristic of language without special training. Another variety of the defect is characterized by all oral consonants and vowels becoming partially nasal and all nasal becoming partially oral, so that they sound stopped-up. These last two defects have a profound effect on speech cadence. The patient seems awkward.

The defect is more common in males than females. The frequency of occurrence of the defect diminishes with age. More females outgrow it than males. In adults its persistence may result from or be due to poor enunciation. However, phoniatric treatment is always indicated whenever the defect is met.

The defect seems to run in families. The enlarged velum seems to be hereditary. The soft-palate development is completed by the eleventh week in the embryo and the condition can be seen at birth, but an absolute prediction of speech defect is impossible. Some individuals will be able to speak clearly with the organic manifestations present, and learn to speak thus clearly without special training, but

others will need a very specialized type of phoniatric training in order to be understood at all.

The prognosis is usually positive. It is complicated by the presence of dyslalias, dysrhythmias, and other dysphoniad symptoms. It is frequently a complication of dysphemia and dyslogia. When accompanied by hardness of hearing, deafness, or paralysis, the prognosis is less definite. Low frequency deafness may make the organic anomaly influence the speech where otherwise it would not operate.

The therapy is sometimes simple and sometimes difficult. One case which appeared no more peculiar or difficult than other cases, was never helped. But almost a hundred severe cases in succession have been treated by training voluntary control of the velar sphincter. The soft palate is probed until resistance stimulates a natural closure of the velar sphincter, with the soft palate appearing to elevate and Passavant's ridge operating posteriorly. The initiation of the treatment is sometimes expedited by elevation of the chin for early exercises.

After sphincter control has been taught, the patient is trained to hear the quality differences when the sphincter is closed and when it is open. He is then given exercises planned to develop this sense of contrast in timbre. After he has learned the art of speaking with the nose closed off for all speech except the three nasal consonants, he is trained to produce the nasal consonants and finally to combine them into rapid speech. The final procedures are concerned with relaxation exercises, so that the voice quality will further develop and not sound stopped-up. Normal voice techniques and vocal hygiene complete the treatment.

REPORT OF CASES

CASE 1.—G., male, age eleven, was brought to the Speech Clinic by the family physican and the mother. G.'s father and parental grandfather had had similar defects of speech when young, but had outgrown them as a result of admonitions to talk slowly. G. had talked this way since starting to talk. The family physician believed that he was slowly improving since he was slowly coming to understand some things that he said. In parturition G. had a birth trauma in forceps delivery. His throat was injured, but at eleven was pronounced normal by a laryngologist. His left eye was injured and was not used for sight. He was in the fifth grade at school and received good grades. G. was a sensitive child and it didn't take much to make the tears come, although there were no demonstrations in the Speech Clinic. He was cooperative. The mother was en rapport with him. He was mechanically inclined.

Speech Examination: His isolated and connected vowels were satisfactory. Initial consonants *s*, *z* and *sh* were weak in breath pressure. *Th* (unvoiced) became f. *Th* (voiced) became *z*. Final unvoiced *th* was unstable, being at times

present and at times absent and at other times substituted for by *s*. Final voiced *th* was omitted. Final *s*, *sh*, and *z*, were weak in breath pressure. *M*, *n* and *ng* were articulated satisfactorily but sounded stopped up, and were sometimes omitted in final position. Sibilants in general were partially nasal or substituted for by nasals. Connected speech sounded both stopped up and nasal.

Diagnosis: Rhinophonia macrostaphyla, with dyslalia complications. This was an organic defect.

Etiology: The disordered voice and speech was the result of an anatomic anomaly and physiological dysfunction. The velum was in close approximation to the epiglottis, which was conducive to nasal speech and in the case of this patient, to a divided oral nasal expiration for utterance.

Therapy: The voice problem was handled by first clearing up the articulatory problems which concomitantly removed the voice defect. Then voice development proceeded along normal lines. The problem was twofold, in addition to which there were two minor articulatory problems. The first task was to devise a method for the completely oral production of the fricative consonants. The second task was to devise a method for the completely nasal production of the nasals which would not in any way conflict with the treatment of the oral sounds. In both cases the adjustment had to be one of velar manipulation for correct articulation.

G. had attempted to speak softly to prevent fricative consonants from sounding nasal. He was instructed to prod his velum with an applicator until he could maintain the resulting tension. With his sphincter thus closed he was instructed to increase breath pressure for all oral sounds.

For the nasals, G. was instructed to face his head toward the ceiling and blow air through his nose. The head movement made the sphincter treatment seem separate for him, and he had no confusion. He then was instructed to hum with the mouth closed (for *m*) and with the mouth open (for *n*). *Ng* was omitted until final *m* and *n* was mastered. He was instructed to gradually lower his head as he hummed.

After he had learned the isolated consonants, he was instructed in using them in words. As he mastered each word his speech standard was raised to include the correct pronunciation of the new word.

Progress: The mother and family physician were instructed in the daily therapy. The mother carried out the training under the guidance of the physician. The articulatory problem was cleared up within a year, but his speech was still unclear on some words. He found it difficult to break old habits on already learned words. He could pronounce any word clearly if he spoke slowly. The prognosis at this time was, of course, that if he continued to give attention to his speech, he would soon have no further trouble.

CASE 2.—K., male, age six years. Mother reported that K. could not be understood and could not pronounce several letters. K. had spoken defectively since he had started to talk. Parental relatives stated that his father had sounded greatly like K. when he started to talk. K. seemed to be a normal boy mentally and emotionally. The medical examination was negative.

Speech Examination: All plosives were nasal and unexploded. *K* and *g* became *t* and *d*. Nasals were satisfactory except *ng* which became *m*. All fricatives were

nasal or omitted. All vowels were nasal. Speech could not be understood except by those who heard him constantly.

Diagnosis: Rhinophonia macrostaphyla, an organic defect. The voice and speech were both nasal. The velum was forward and long, and in close approximation to the epiglottis. There was an organic articulatory problem also as a result of the structure of the palate. The hard palate was fused anteriorly but separated one-fourth inch posteriorly. The bony cleft was sealed by membrane. The hard palate was high and vaulted.

Etiology: The voice disorder was the result of the velar structure which caused the expiration intended for voice and speech to pass through the nose, leaving the language articulated but not spoken. Emotional vocal expression was highly developed. Any linguistic expression seemed accidental and was due to an occasional partial oral utterance. In any event it was as soft as a whisper, although the voice was loud.

Therapy: The general problem was merely to train K. to avoid any nasality on any sound which should be oral by a rather special articulatory stance made necessary by the anatomical situation. There was a special problem in regard to *k*, *g*, and *ng*, in that they had to be produced gutterally as a result of the anatomic structure of the palate which had to take into consideration that the glossopalatal relation was distal, whereas the glossovelar relation was proximal; it was found that this substitute articulation was phonemic with *k*, *g*, and *ng* in English and not too great an acoustic divergence. The mother was trained to drill K., since he was brought to the Speech Clinic only five times and lived out of state. K. was trained to close the velar sphincter by probing. He was trained to blow through his mouth (extinguish matches, etc.). He was then taught "voice placement" by having him speak when his sphincter was closed and his tongue was moved forward as a body. He was then taught to articulate phonetic consonants through his mouth, working first on plosives and then fricatives. The liquids (*l* and *r*) were omitted from the training until he had learned to speak orally on all the rest of his speech. They were omitted from his speech simply as a matter of arhota- and alambdalalia prolongata. He was instructed to move his tongue more backward than upward for *k*, *g*, and *ng*.

Progress: At the end of six months, K.'s speech was again examined. K. was in the first grade of school as he had been advised, but was still at some disadvantage. His nasals were still satisfactory. *R* and *l* were still omitted. K. was still drilling on some phonetic sounds: *s*, *sh*, *k*, *g*, and *ng*. K. was still drilling on words and sentences containing the following consonants: *p*, *b*, *t*, *d*, *f*, *v*, *w*, *wh*, *j*, *z*, *zh*, and the *th*'s. His voice was satisfactory. The mother seemed to understand the the problem of diagnostic speech re-education and by the end of eighteen months he was speaking satisfactorily, whereas at the end of six, he could be understood in long recitations only if he spoke slowly, applying all his speech control.

Paget's[6] exercise was of considerable convenience in these cases. It is a poem without any nasal consonants. It was used for connected utterance when the patients first started to enunciate phonetic continuums with the velar sphincter closed:

"Hard by the shores of far Brazil
We rode for pleasure years ago,
Led forward ever by the will
To brave each risk, to fight each foe."

The counterpart of Paget's exercise contains nasal consonants and is often valuable for comparison in diagnoses.

"No roaming more. In time nigh spent
Mere strength grown faint, new prudence strong;
And now from home my mien unbent
Must amble near my mate—in song."

201 WENTWORTH HALL.

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LII

TRACHEAL STENOSIS FROM ROENTGEN THERAPY*

Louis H. Clerf, M.D.
AND
F. Johnson Putney, M.D.

Philadelphia

Complications resulting from intense irradiation in the treatment of carcinoma of the larynx are well recognized by laryngologists and radiologists. While there may be justification for these in advanced or otherwise hopeless conditions, there is grave question concerning the desirability of employing such measures in great amount as a form of elective treatment for conditions compatible with life. Recent experiences have served to emphasize these hazards in the irradiation treatment of exophthalmic goitre.

According to Aschoff,[1] the cells of the epidermis, blood building and vascular tissues, glandular organs and other structures capable of active physiologic cell regeneration suffer from the effects of the roentgen ray while fibrillar elements withstand irradiation to a greater degree. The primary effect of the roentgen ray on tissue is not destructive or disabling. This reaction appears in from ten to twenty days after radiation and usually does not interfere with the patient's activities. However, when treatments are frequently repeated or given over long periods of time, there occur pronounced alterations in the vascular endothelium with obliterative changes in the vessels resulting from trophic reactions. This is designated as the late reaction and may occur within a few weeks after the termination of the treatment or it may be delayed for one or more years. This often is associated with extensive tissue changes. The muscles undergo degeneration and are replaced by fibrous tissues, the tissues are matted together and lines of cleavage are obliterated. Cartilage may undergo complete destruction. Marschik has given the designation "perichondritis radiotherapeutica" to the perichondritis and necrosis of the laryngeal cartilages. In addition to the trophic changes, Lowen[2] has directed attention to decreased tissue resistance to bacterial infection resulting from injury of the blood forming organs.

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From the Bronchoscopic Clinic, Jefferson Hospital, Philadelphia, Pa.

During the past two decades there have been recorded many reports of roentgen ray injury to the larynx and neck. A majority of those treated were case of carcinoma and lymphoma. In Iglauer's[2] report on x-ray injury to the larynx, he found 27 cases in the literature and added a personal case. Thirteen of the 27, 48 per cent, proved fatal. In 1927, one of us[4] reported two instances of late irradiation reaction occuring five and nine years, respectively, following intensive roentgen treatment for exophthalmic goitre. Stenosis of the larynx with extensive perichondritis and gangrene of the laryngeal cartilages had occurred. One of these patients died from bronchopneumonia. The other patient recovered following a tracheotomy performed for obstructive laryngeal dyspnea, but a considerable portion of the hyoid bone, the cricoid and thyroid cartilages and several tracheal rings were destroyed. The wound never completely healed. In both of these patients the stenotic lesion was limited to the larynx without narrowing of the tracheal lumen.

Kilduffe[5] reported a similar fatal case occurring five years after a prolonged course of roentgen treatment for exophthalmic goitre. There was gangrenous destruction of the larynx and contiguous portion of the base of the tongue. Gross obstruction of the trachea was not found.

In a case reported by Posey,[6] the patient had previously received an average of two roentgen treatments weekly for a period of fifteen months for exophthalmic goitre. At the end of this time she developed severe dyspnea and died, although tracheotomy was performed. There was found a tracheo-esophageal fistula.

In none of the reported cases were there available data indicating the amount of roentgen therapy given or the method of its employment. The extensive tissue destruction would indicate that a considerable amount had been given. From the records it was apparent that the periods of treatment were prolonged.

The following cases are of interest not only from the standpoint of having received irradiation therapy in large amounts for exophthalmic goitre, but principally because of the occurrence of delayed disabling tracheal lesions without laryngeal involvement.

REPORT OF CASES

CASE 1.—A woman, aged 48 years, was referred to the Bronchoscopic Clinic, Jefferson Hospital, by Dr. J. E. Womack because of severe dyspnea, cough with expectoration of blood-tinged secretion and weight loss. Eight years previously a diagnosis of exophthalmic goitre had been made and intensive roentgen therapy was instituted. Following completion of the treatment, which required about one year, the patient noted some dyspnea on exertion. This became slowly but progressively

Fig. 1. Drawing of granuloma found partially occluding the lumen of the trachea. The surface was smooth. The point of attachment was anterior.—(Drawn by E. G. Keenan.)

worse, so that ultimately she could not assume a recumbent posture when sleeping and was unable to perform any of her household duties. There was increasing weight loss.

On admission to the clinic there were observed evidences of marked inspiratory dyspnea. By mirror laryngoscopy the larynx appeared normal. The skin over the lower neck anteriorly was atrophic and grossly and marked telangiectasis was present. The chest was essentially negative both by roentgen and physical examination. At bronchoscopy performed under local anesthesia there was observed almost complete obliteration of the tracheal lumen at the level of the second ring. The stenosis was due to narrowing of the lumen of the trachea and the presence of a granulomatous mass and inspissated secretion. The tracheal lumen, crescentic in shape, was observed posterior to the granuloma (Fig. 1). Tracheotomy was performed, the tube being inserted below the point of stenosis. Later the granuloma was removed bronchoscopically. The point of attachment was to the anterior tracheal wall. At this point the diameter of the trachea was about one centimeter. The diameter of the normal trachea above the stenosis was slightly more than two centimeters, indicating that there was definite narrowing of the trachea itself. The granuloma was one-half centimeter in diameter. It consisted of a loose connective tissue center containing many large vessels and covered by stratified squamous epithelium. The histologic diagnosis by Dr. B. L. Crawford was fibroangioma. Additional bronchoscopy and bouginage were required, but the patient ultimately was decannulated and has been able to resume a fairly normal life. Insufficient time has elapsed to determine whether the stenosis may increase, the granuloma recur or further atrophic or destructive changes develop.

Case 2.—A woman, aged 31 years, waitress by occupation, was referred by Dr. C. T. Towson. She had received a course of deep roentgen therapy for exophthalmic goitre six years previously. Approximately three years following this there was noticed shortness of breath on exertion which became progressively more marked; in addition, there were frequent paroxysms of cough with expectoration of mucopurulent sputum and occasional fetid greenish crusts. The nose, throat and

larynx were essentially negative. By mirror laryngoscopy one could see crust adherent to the tracheal mucosa immediately below the vocal cords. There was present moderate telangiectasis of the skin of the front of the neck with some scarring. The chest was negative by physical examination and roentgen study. The Wassermann test was negative.

By tracheoscopy there was found marked narrowing of the lumen of the trachea beginning at the level of the third tracheal ring and extending downward for a distance of about one centimeter. The mucosa was atrophic in appearance and exhibited small granular areas that bled readily when touched. Crusting of secretions also was observed. The lumen at the point of stenosis barely admitted a 7 mm. bronchoscope. The tracheal lumen therefore was not greater than one centimeter in diameter.

Treatments are being directed towards removal of crusts and epithelialization of the tracheal mucosa. Dyspnea on exertion still persists, especially when there is crusting of secretions. Cough is particularly troublesome. The larynx and the voice are normal.

The history of irradiation therapy, the occurrence of marked telangiectasis of the skin over the irradiated areas and the presence of atrophic changes of the tracheal mucosa with stenosis of the lumen of the trachea at this same level would indicate that these unfavorable after-effects are of roentgen-ray origin. This view may be further corroborated by the absence of systemic disease, recent infection or injury to the affected structures. Whether these reactions are due to excessive dosage, inadequate filtration, individual susceptibility or cumulative effects are problems for further investigation. It is of interest to note that the larynx sustained no injury.

In conclusion, it must be emphasized that powerful therapeutic agents are potential sources of danger. If irradiation has a place in the treatment of exophthalmic goitre, can proper precautions be exercised to avoid trophic reactions which are disabling and may terminate fatally?

1530 Locust Street.

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LIII

THE UNDERLYING FACTORS CONCERNED IN OTITIC HYDROCEPHALUS*

HENRY L. WILLIAMS, M.D.†

ROCHESTER, MINN.

With the increasing number of cures of septic otitic meningitis, both through the agency of sulphanilamide and other methods, we may expect hydrocephalus to become a problem confronting the otologist more and more frequently. In reviewing the literature of so-called otitic hydrocephalus, it is at once obvious that it is not an isolated pathologic phenomenon associated with disease of the ear, but an example of that great group of intracranial hypertensions on an inflammatory basis considered in the literature under the various names of serous meningitis, meningitis sympathica, ventricular meningitis, acquired hydrocephalus, cerebral pseudo-tumors, pseudo-brain abscess or otogenous encephalitis. It is impossible to draw a definite line of demarcation between the acute, the subacute, the recurrent cases of otitic hydrocephalus or aseptic meningitis on the one hand and chronic hydrocephalus on the other. The one grades into the other, both in symptomatology and pathology. It therefore becomes necessary, in fulfilling the requirements imposed by the title of this paper, to consider briefly the anatomy, physiology and pathology which might have a bearing upon the production and absorption of the cerebrospinal fluid, and any alterations in any or all of them tending to produce a state of intracranial hypertension.

DEFINITION

The term "otitic hydrocephalus" was suggested by Symonds[1] "because the term implies no active process of inflammation and since it is free from the qualifications internal or external, it includes conditions in which fluid is within both the ventricles and the subarachnoid space." Since several authors have shown that a similar clinical syndrome can be produced by infections in the paranasal sinuses or even by a general toxemia which may or may not be associated with an otitis, and that the limits between "active" and "non-active" in-

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†Section on Otolaryngology and Rhinology, The Mayo Clinic, Rochester, Minn.

flammatory processes cannot be clearly drawn, this term is obviously not completely accurate. It has, however, the advantage of terseness and it suggests the association with otitic disease which is usually present. These advantages will cause it to be retained in the memory while serving to put the otologist on the alert for its presence, and for these reasons it seems to be the most generally useful term.

Passot[2] preferred the term "aseptic meningeal states of otitic origin." He considered the aseptic meningeal process under two headings. The first, in which there was a simple outpouring of cerebrospinal fluid without any cellular elements or albumin, he termed the hypertensive state. The second, with an increase of cellular elements and albumin or an increase in albumin alone, he termed the "true aseptic meningitis, serous or purulent." In regard to the first division he stated, "We include under the name hypertensive states a class of meningeal phenomena characterized by an enormous excess of cerebrospinal fluid in the arachnoid and ventricular cavities; a clear fluid without cellular elements and without albumin; appearing in infancy or in youth in the course of an otitis, without a destructive osteitis of the mastoid bone; without inflammatory changes in the dura or disease of the labyrinth or alarming clinical symptoms and in which the draining off of the excess cerebrospinal fluid produces an immediate and definite relief." Passot further divided the acute hypertensive state into meningeal hydropsy and acute internal hydrocephalus and found that the two were usually co-existent. He pointed out, however, that ventricular hypertension could exist alone and then owed its existence to an occlusion of the foramina of Mangendie or Luschka or of the aqueduct of Sylvius, either due to a congenital or chronic lesion or to edema of the ependyma and underlying brain tissue. He recognized an acute and a subacute form of acute ventricular hydrocephalus. Symonds,[1] in his paper on otitic hydrocephalus, accepted Passot's classification. It seems to comprehend adequately both the pathologic findings and the possible symptom complexes which may appear in this condition and seems to be the best detailed classification of the subject.

THE LITERATURE

A review of those articles in the literature which illustrate the gradually developing conception of otitic hydrocephalus and its differentiation from septic meningitis may serve to give a broader background for a survey of the problems involved. The recent literature on the subject, especially that published in the United States, is considered in more detail.

The ancients made no distinction between the several inflammatory affections of the intracranial organs, but comprehended them all in one disease which they called "frenzy." Morgagni, however, showed that the membranes of the brain were the parts generally involved. In 1768, Robert Whytt[3] distinguished between acute hydrocephalus of infancy and the so-called congenital hydrocephalus. Gólis,[4] in 1815, wrote the first monograph on hydrocephalus or "inflammatory water in the head." These writers, of course, made no distinction between inflammatory aseptic hydrocephalus, tuberculous, cerebrospinal or other forms of meningitis or hydrocephalus from cyst or tumor. It is interesting to note that in his autopsy material Gólis[4] clearly described three cases of meningitis secondary to necrosis of the apex of the petrous pyramid, possibly the first recognizable descriptions of this lesion in the literature. Barthez and Rilliez,[5] in 1843, distinguished tuberculous from other inflammatory forms of intracranial hypertension. Grisolle,[6] in 1850, observed that in acute meningitis the disease could advance into the ventricles. Lambl,[7] in 1860, suggested the relationship which frequently exists between acute hydrocephalus and basal meningitis and thought primary acute hydrocephalus was very rare. Hanot and Joffroy,[8] in 1873, noted a closure of the fourth ventricle following a meningitis, with death from symptoms suggesting acute hydrocephalus a month after the cure of the meningitis. Huguenin,[9] in 1878, described subependymal perivascular infiltration in a case with intraventricular suppuration. Wernecke,[10] in 1881, described ependymal inflammation in the course of inflammations of the arachnoid sac, including thickening, edema, friability and infiltration with pus. Barthez and Sanné,[11] in 1884, distinguished two sorts of liquids in hydrocephalus, the one completely clear without or with only a trace of albumin, the other containing leucocytes and an excess of albumin. Wiederhofer and Frühewald,[12] in 1885, described primary inflammation of the ventricles in early infancy without meningitis. Wälter,[13] in 1888, described inflammatory changes in the ependyma of a case of chronic hydrocephalus which led to death. From this time until 1893, when Quincke[14] published his paper on meningitis serosa, the literature contains many reports of similar cases. In 1891, Quincke[15] suggested the use of lumbar puncture (apparently done for the first time on the human) as a method of treatment for hypertensive meningeal states. In 1896[16] he described a clinical syndrome of serous meningitis, basing his paper on fourteen personal cases, to which he added those of Annuske, Oppenheim, Eichhorst, Plehn and Socin. The cases, however, are quite different. In some there seems to have been an old hydrocephalus with exacerbation and in others tuberculosis appears

to have been suspected. The descriptions of the pathology are based on gross examination alone. He mentioned, particularly, thickening or a granular condition of the ependyma. In this paper, Quincke[16] suggested that an angioneurotic mechanism produced the excessive cerebrospinal fluid in those cases which responded best to lumbar puncture.

Following Quincke the reports of serous meningitis were very numerous. In the United States, Joslin,[17] in 1900, reported a case of internal hydrocephalus following meningitis. Byron Bramwell (1894), Hammerschlag (1900), Beck, Licene and Bourgeois (1902), Broca, together with Voss (1902), Joel, Kritschmann, Oppenheim, Lucae, Waldvogel, Müller, Bruger, Hegener and Hansen, who are all cited by Oppenheim[18] in 1913, published reports of cases in which intracranial hypertension was associated with otitic disease. Herzfeld,[19] in 1905, reported a case of serous meningo-encephalitis associated with disease of the paranasal sinuses. Beck,[20] in 1903, performed autopsies on five cases which were diagnosed as serous meningitis. In the three cases which were examined histologically, he reported the predominant lesions to be leucocytic infiltration, especially around the vessels in the ventricular walls. In 1910 the exhaustive thesis of Merle[21] appeared. He endeavored to show that almost all cases of hydrocephalus were associated with lesions of the ependyma, but laid special stress upon subependymal perivascular infiltration. In 1913, Passot[2] published his thesis on otitic hydrocephalus in which he suggested "practically aseptic" meningeal processes as the most useful descriptive term which could be applied to the condition under consideration.

Following this paper, there was a sudden cessation of reports in the literature, whether due to infrequency of the symptom complex, lack of interest in the subject, or the effects of the World War, it is difficult to state. When reports begin to reappear, the previous literature on the condition seems to have been largely lost sight of for a time. Borries,[22] in 1921, developed the theory of an otogenic encephalitis differing from that form of encephalitis which leads to the formation of abscess. He reported two cases which suggest the possible presence of an undiagnosed ventricular hydrocephalus. Holger Mygind,[23] in 1922, in association with mastoiditis, reported thirty-eight cases of benign purulent meningitis, and fifteen cases of "collateral meningitis" in which the cerebrospinal fluid was clear. In several of these cases the presence of a ventricular hydrocephalus is suggested by the symptoms presented. Strauss,[24] in 1926, published a valuable paper on meningitis sympathica in which he stated

that acute suppurative conditions in either the ear or sinuses frequently cause a reaction in the cerebrospinal fluid, characterized by an increase in the protein and cells in the fluid. These changes in the fluid were associated with signs of meningeal irritation. This reaction had received the name of meningitis sympathica from Plaut, Rehm and Schottmüller.[25] He advised that when this syndrome appeared, steps should be immediately taken to eliminate the original focus of infection. Adson,[26] in 1924, reported three case of "pseudo-brain abscess" associated with mastoid disease. Although exploration for abscess of the brain was done in two cases, the ventricle was not tapped and the patients made a slow recovery. Adson expressed the opinion that the symptoms were attributable to localized encephalitis, although it seems possible from the case histories that a hydrocephalus was present. He regarded the possibility of localized encephalitis as a reason for delaying exploration in cases of suspected abscess of the brain. Yerger,[27] in 1925, reported three cases of "acute toxic meningo-encephalitis" in association with mastoiditis, and one case in which the condition was in association with acute frontal sinusitis. Key-Åberg,[28] in 1926, reported a case in which paralysis of the left side of the face and paresis of the left arm developed after mastoiditis on the right side. On incising the dura, cerebrospinal fluid gushed out under great pressure. It is possible that Key-Åberg was dealing with a circumscribed subdural collection of fluid rather than with a generalized hydrocephalus. Symonds,[29] in 1927, reported three cases in which a diagnosis of abscess of the brain was made. Two cases were explored for brain abscess with negative results. He was of the opinion that a "presuppurative" stage of abscess of the brain was present in these cases, in which there was engorgement, exudation and swelling without necrosis. In a later paper,[1] 1931, he altered his conception of the pathologic picture to agree with that of Passot. O. Voss,[30] in 1928, reported two cases in which he termed the condition "acute toxic encephalitis in association with acute influenzal otitis mdia" and two cases of "encephalitis serosa without any sign of serous meningitis." Cairns,[31] in 1930, reported a case of serous meningitis associated with mastoiditis simulating cerebellar abscess. He pointed out the dangers of a serious affection of the vision if these cases were allowed to continue. Symonds,[1] in 1931, reported three additional cases suggesting brain abscess in association with mastoid disease. In one, operation was performed, with the discovery of internal hydrocephalus; in the two others the discovery of normal cerebrospinal fluid led to the correct diagnosis of "otitic hydrocephalus." In this paper Symonds suggested the designation "otitic hydrocephalus" for the condition under consider-

ation. Carmack,[32] in 1933, reported a case of subacute hydrocephalus in which the condition was similar to that observed in association with otitic disease which was attributable to suppuration in the sphenoid sinus. Garland and Seed,[33] in 1933, reported two cases, in one of which definite meningitis with organisms and increase in concentration of protein in the spinal fluid, followed the earlier development of a serous meningitis. The patient made an eventual recovery. Smith,[34] in 1934, reported a case in which primary signs suggesting otitic hydrocephalus were followed by definite suppurative meningitis with organisms present in the cerebrospinal fluid. Nielsen and Courville,[35] in 1934, attempted classification of the cases of "pseudo-abscess" of the brain into five groups, with the first group divided into two subgroups. Since Nielsen and Courville could conceive of no integrating pathologic basis for any of their groups, the only thing in common being the difficulty in distinguishing the condition from abscess of the brain, the "classification" is little more than a series of isolated case histories. One case was reported to illustrate each of the six types.

Bourgeois,[36] in 1935, compiled a monograph on otitic hydrocephalus with sixty-one case histories in detail, two of his own, the remainder collected from the literature. He conceives the cause of otitic hydrocephalus to be "d'accidents vaso-moteurs purs," and because of this theory he believes the underlying pathology is either nonexistent or merely edema. His classification consisted of dividing hydrocephalus into multiple types, emphasizing neither their differences nor similarities, and confusing rather than clarifying. He added nothing new to diagnosis or treatment. Rabbiner,[37] in 1935, reported a case of pseudo-abscess of the brain associated with mastoiditis. Ersner and Myers,[38] in 1936, reported five cases of "otitic hydrocephalus." They suggest the hypothesis that circulatory disturbances about the petrous pyramid, especially obliteration of the lateral sinus, are an important factor in the production of this condition. They felt that it was unnecessary to consider the literature in regard to this condition, yet one is struck with the thought that had they done so this hypothesis would not have been presented. Moulonguet and Demaldent,[39] in 1936, reported the case of a patient with serous meningitis in association with mastoiditis who died of septicemia. At autopsy they were able to find no gross changes in the meninges or ependyma. Shambaugh,[40] in 1937, reported five cases of meningitis of otitic and nasal origin. In four of these cases there was no growth of organisms on culture or smear of the spinal fluid and they were the type usually classified as meningitis sympathica or aseptic meningitis. Adam and Connal,[41] in 1937, reported

nine consecutive cases of purulent meningitis with recovery. Five of these cases were without organisms and from the history and findings correspond to the group of aseptic meningitis associated with mastoiditis. Bradbeer and Gibson,[42] in 1937, reported a case of otitic hydrocephalus with papilledema and apparently a communicating hydrocephalus with a cerebrospinal fluid pressure of 300 mm. (of water). Repeated punctures gradually reduced the papilledema. In a paper entitled "Otitic Hydrocephalus," I[43] presented four cases, three illustrating an internal hydrocephalus and one a circumscribed external hydrocephalus associated with an internal hydrocephalus. The rapid recovery of those patients in which ventricular drainage was done was stressed. DeOcampo,[44] also in 1937, presented two cases of serous meningitis with a detailed description of the preoperative examination and operative findings. He discussed logically the cause and treatment of these conditions. Ramadier[45] presented one case of internal hydrocephalus and one case of collateral meningoencephalitis which were only differentiated from brain abscess at operation. He discussed the proper operative procedure in similar cases.

ANATOMY

The brain and spinal cord are enclosed in a rigid encasement of bone. Underlying this and loosely attached to the surrounding bone at most points is the fibrous inelastic envelope of the dura mater. Under this are the loose meshes of the pia-arachnoid through which the larger cerebral arteries from the carotids and vertebrals run. According to Sepp,[46] the subdivision of the pia-arachnoid into two membranes is not justifiable. He therefore calls the endothelial cell container of the cerebrospinal fluid the arachnoid cavity, the external wall being connected with the dura mater and the inner wall with the glial tissue. As the large arteries divide and subdivide, they finally pierce the pia mater, carrying with them an invagination of this delicate membrane. Weed[47] showed that these vessels have a double perivascular sleeve, a visceral lining of arachnoid and a parietal lining of pia. According to Sepp,[46] these prolongations of the arachnoid sac cover the finest ramifications of the vessels except the capillaries. Mott[48] thought the perivascular and perineuronic spaces are continuous and believed that the cerebrospinal fluid surrounds the neurons and plays the part of intercellular fluid to the central nervous system. The extravascular and extraneural spaces form the main body of the arachnoid cavity and can be roughly differentiated from the perivascular and perineural portion. In the former extravascular spaces the inner wall (the pia mater) follows all the irregularities of the brain surface; the external wall (the arachnoid) passes over

these structures leaving larger or smaller spaces (the subarachnoid spaces), the larger of which form the various cisternae. The cisterna magna or cisterna-cerebello medularis is located between the cerebellum and the posterior part of the medulla oblongata; the cisterna basalis in the region of the circle of Willis, the cisterna chiasmaticus in the region of the optic chiasm, and the cisternae lateralis and medialis, the pontile cisterns, stressed by Eagleton[19] lie in relation to the petrosae. The ventricular system is also lined with the cells of the internal wall (the pia) of the arachnoid sac. The lateral ventricles are connected with the third ventricle and through its agency with each other through the foramina of Monro. The third joins the fourth ventricle through the aqueduct of Sylvius, and the fourth ventricle communicates through the foramen of Magendie, and the foramina of Luschka with the cisterna magna.

The capillaries of the ventricular walls give birth to the deep cerebral veins of the system of Galen. The three principal vessels are the choroid veins, the vein of the corpus striatum and the vein of the septum pellucidum. Their union in the region of the foramen of Monro produces on each side the internal cerebral vein or small vein of Galen. These veins, running posteriorly toward the corpora quadrigemina, receive, among other collaterals, the veins of the inferior (temporal) and posterior (occipital) ventricular horns. They finally unite to give a single trunk the great vein of Galen which, joining with the stright sinus, enters into the torcular Hierophili. The cerebral and spinal nerve roots are also covered by the endothelium of the inner wall of the arachnoid sac. In the spinal canal this membrane, according to Sepp,[16] accompanies the nerves until they reach the intervertebral ganglia, where it joins with the dura mater, cutting off the archnoid cavity from the lymphatic spaces of the peripheral nervous system. The cranial arachnoid sac continues outside the cranial cavity along three nerve pathways: the filia olifactoria of the olfactory nerves; the optic nerves along which the arachnoid sac is prolonged to the eye grounds; and the nerve pathways of the internal ear, the whole perilymphatic system being a direct continuation of the arachnoid cavity. The whole arachnoid cavity, the ventricles, the cerebrospinal subarachnoid spaces, the cisternae, the perivascular, perineural and pericellular spaces constitute a unit.

HYDRODYNAMICS

It can be readily appreciated that anything tending to interfere with any of the factors which maintain the normal balance of forces within the craniovertebral cavity may tend to produce a hydro-

cephalus. According to Loman and Myerson,[50] the physics of the cranio-vertebral cavity is unique. Although the brain and spinal cord are invested in a rigid encasement of bone, it should be stressed that the conditions within the cavity only approximate a closed box system. This becomes immediately apparent when one considers the fact that the blood and cerebrospinal fluid which leave the cranial and vertebral cavities have many exits which communicate with the spaces outside these cavities. Weed and Hughson[51] demonstrated that within tested physiologic limits the ordinary physical laws of a closed box may be applied to the cranium, however. Enclosed within the craniovertebral cavity are the practically incompressible brain and spinal cord invested by the thick outer dura and the thin inner pia arachnoid. Since these membranes are inelastic, Loman and Myerson[50] thought they can play but an insignificant role in the cerebrospinal fluid pressure. Weed and Flexner,[52, 53] however, found two factors, the elasticity of the membranes and the elasticity of the intradural blood vessels to be closely related to pressure changes in the cerebrospinal fluid. While the meningeal elasticity is structurally determined, the elasticity of the vessels is largely under the control of the vasomotor nervous system. These findings do much to explain the postural differences in the pressure of the cerebrospinal fluid found by several authors. Loman and Myerson[50] stated that while there is little potential intradural space within the cranial cavity, the spinal cord, on the other hand, is surrounded by a loosely applied dura, so that the spinal cavity is relatively capacious as compared with the volume of its contents. Thus, under many conditions the spinal subarachnoid space may act as an emergency reservoir when cerebrospinal fluid is expressed from the cranial subarachnoid spaces. This is mainly made possible by the presence of the many thin-walled, easily compressible spinal veins. Loman and Myerson[50] also stated that in considering the reciprocal relationship of the venous and cerebrospinal fluid pressure, it should be stressed that it is the thin-walled cerebral veins which are important and not the cranial sinuses. Since these latter channels are enclosed in thick dural membranes, they can exert but little influence on the cerebrospinal fluid pressure, nor can they in turn be influenced by the latter pressure. In contrast to this the cerebral veins having walls which are compressible and distensible, can readily communicate their pressure to the cerebrospinal fluid, and furthermore, the cerebrospinal fluid pressure can readily be communicated to the veins. From their experiments upon the effect of alterations in posture and drugs on the craniovertebral dynamics, Loman and Myerson[50] concluded that the cerebrospinal fluid pressure directly follows either a change in venous pressure or a change

in caliber of the cerebral vessels. Thus a rise in arterial pressure does not affect the cerebrospinal fluid pressure unless the venous pressure as a result becomes increased. Sepp[46] was of the opinion that the pacchionian bodies furnished compressible areas, compensating at least for sudden and minor increases in cerebrospinal fluid pressure. Tzanck and Renault[51] showed that the cerebrospinal fluid pressure is usually about 12 to 14 cm. of water and corresponded to the venous pressure. They presented several cases in which elevated venous pressure was reflected in an equally elevated pressure of cerebrospinal fluid. Weed and Hughson[55] concluded that normally the pressure is from 5 to 50 mm. of water above the venous pressure in the saggital sinus. Jackson[56] pointed out that the pressure is maintained by the secretion of the choroid plexuses, and that normally the total quantity of cerebrospinal fluid is from 120 to 150 cc. Anton[57] thought that the choroid plexus acted as a filter to prevent poisonous substances from the blood entering the cerebrospinal fluid, and that the autonomic nervous system controlled the pressure through its control of the blood pressure and of the filtration processes of the choroid plexus. Hart[58] found that under normal conditions there is a decrease in the amount of cerebrospinal fluid secreted in the early morning and afternoon, and an increase in the amount secreted in the forenoon and evening. The practical cessation in the formation of cerebrospinal fluid for the few hours after midnight, Hart[58] found most striking. He found that irritants introduced into the ventricles will give a marked increase in the formation of fluid without regard to day or night.

Weed and Hughson[55, 59] demonstrated that changes in volume and concentration of the blood caused changes in pressures of the cerebrospinal fluid and of the circulatory system. Large amounts of Ringer's solution given intravenously induced a transient rise in the brachial vein and cerebrospinal fluid pressures, whereas the arterial pressure went down at once during the injection and remained slightly subnormal. Distilled water given intravenously provoked a sustained rise in the cerebrospinal fluid pressure and a rise of smaller degree and shorter duration in the brachial vein. Hypertonic solutions introduced into the vein induced a marked and prolonged decrease in the cerebrospinal fluid pressure which was associated with an initial rise and a subsequent fall in the venous pressure. The inference drawn was that alteration in the cerebrospinal fluid pressure produced by changes in the volume and concentration of the blood seemed to occur independently of changes in the systemic arterial and venous pressures. The cerebrospinal fluid pressure was shown, however, to depend in larger measure upon cerebral venous rather

than upon cerebral arterial pressure. Weed and Hughson[55] found marked correspondence between cerebrospinal fluid pressure and pressure in the brachial vein. Sepp[46] stated that additional dynamic causes actuating the circulation of the cerebrospinal fluid are an increase in brain volume during cardiac systole and a decrease in brain volume during cardiac diastole and by venous dilatation during inspiration and collapse during expiration, thus tending to produce lowered venous pressure in the dural sinuses. Weigeldt[60] was of the opinion that movements of the body, especially bowing of the head, insofar as they cause constant changes in the intracranial and intraspinal pressures are also dynamic forces in the circulation of the cerebrospinal fluid.

PRODUCTION OF CEREBROSPINAL FLUID

The production of the cerebrospinal fluid has been assigned by most authors to the choroid plexus. The voluminous literature on the subject has been summarized by Katzenelbogen,[61] who stated that the data reviewed present definite evidence that the main source of the cerebrospinal fluid formation lies in the choroid plexus. The demonstration of differences in the constitution of the ventricular and subarachnoid fluids on the one hand, and the findings pointing to diffusion of certain substances from the brain vessels into the peri-advential spaces on the other, suggest that the extraventricular cerebrospinal tissues take a certain part in the formation of the cerebrospinal fluid. According to Katzenelbogen,[61] the theory of secretion, which has been linked by most investigators with the idea that the choroid plexuses present the main source of production of the cerebrospinal fluid, is grounded on two factors: (1) the histology of the plexuses, and (2) the pharmacodynamic effect of certain drugs (reputed to be glandular stimulants) on their structure and on the rate of cerebrospinal fluid production. The opponents of this theory are of the opinion that the would-be histologic secretory features in the epithelium of the choroid plexuses are artificial postmortem formations and that the apparent increased output of cerebrospinal fluid under the influence of glandular stimulants is caused not by overproduction but by hemodynamic reactions in the cerebrospinal cavity. On the other hand, the proponents of the theory of dialysis base their conviction on the similarity between an artificial blood dialysate and that of the cerebrospinal fluid. This theory seems to be unsatisfactory to Katzenelbogen,[61] not only because comparative physico-chemical studies of blood dialysis in vitro and of the corresponding cerebrospinal fluids have not reached altogether corroborating results, but mainly because one is not justified in assuming that the reactions observed in vitro are similar to those observed

in the living organism. There are, moreover, evidences of spontaneous changes in the function of the barrier between blood and cerebrospinal fluid, which suggest a biologic functioning manifesting itself by physico-chemical reactions. If neither secretion or dialysis alone can be applied to the mechanism of cerebrospinal fluid formation, then the term physiologic or biologic permeability appears to present an adequate expression of a viewpoint that would be a compromise between the concept of secretion and that of dialysis. Kubie,[62] in a summary of his investigations done up to 1934, stated that he had added these further facts to the classical demonstration of Weed: (1) "Under normal circumstances (i. e., with an intracranial pressure that is higher than atmospheric pressure), practically all the cerebrospinal fluid is formed in the choroid plexus. On the bases of Starling's[63] fundamental studies of the method of lymph formation, it is possible to prove that under conditions of normal or increased intracranial pressure, all fluid which transudes through the arterial end of the capillaries, throughout the central nervous system must be reabsorbed at the venous end of the capillaries, and therefore is not added to the main lake of cerebrospinal fluid which lies in the subarachnoid space. (2) When intracranial pressure is dropped to atmospheric pressure and kept there by continuous drainage, the cerebrospinal fluid is formed not only at the choroid plexus but by transudation through all of the small capillary vessels of the central nervous system. Under these conditions—that is, with reduced intracranial pressure—reabsorption at the venous end of the capillary system is reduced, and therefore the capillary transudate is not all reabsorbed but remains in the perivascular and perineuronal spaces and ultimately adds itself to the lake of the cerebrospinal fluid in the subarachnoid space. (3) Under these conditions the fluid which is formed as a capillary transudate will move slowly along the perivascular spaces to emerge finally in the subarachnoid space. (4) The rate of formation of fluid as a capillary transudate can be remarkably accelerated by lowering the osmotic pressure of the blood, if this fluid is allowed to escape freely into the subarachnoid space by maintaining continuous drainage throughout the process. (5) If free escape is allowed for the newly formed cerebrospinal fluid during the period in which the osmotic pressure of the blood is being lowered, there will be no material increase in intracranial pressure and no diffuse hydration of the substance of the cerebrospinal nervous system." This work clearly demonstrates the effect in producing a hydrocephalus which lowering the osmotic pressure of the blood might have, and further suggests the great value of supplying patients with intracranial lesions with sufficient salt.

The Hemato-Encephalic Barrier: Stern and Gauthier,[64] in 1921, established the existence of a barrier which they called the "barriére hemato-encéphalique." They showed that all substances introduced into the general circulation could not be found in the cerebrospinal fluid, but by contrary, all substances introduced into the cerebrospinal fluid after a greater or lesser time could be found in the general circulation. Stern and Rapaport[65] have shown, in studying barrier permeability for certain substances introduced into the general circulation, that it can be assumed that in so far as the anatomic location of the barrier is concerned, an important part of it lies in the epithelium of the choroid plexuses and in the vascular endothelium. These studies show that the epithelium of the choroid plexus acts as a permeable membrane in one direction but as a biologic membrane in the other. Stern and Peyrot[66] showed that in animals born fully mature, ferrocyanide of sodium would not pass the barrière hemato-encéphalique, but that before maturity (intra-uterine) and in animals not fully matured at birth it will pass until full maturity is reached. These facts suggest an explanation for the greater frequency of the appearance of toxic substances in the cerebrospinal fluid of children producing states of intracranial hypertension. Stern, Slatowierow and Belkina[67] were unable to demonstrate any effect of the autonomic nervous system upon the hemato-encephalic barrier.

THE CIRCULATION OF THE CEREBROSPINAL FLUID

According to Key and Retzius,[68] Cathelin was the first to suggest a circulation of the cerebrospinal fluid. The validity of this theory was denied by Walter,[69] Solomon, Thompson and Pfeiffer,[70] Sachs, Wilkens and Sams[71] and others, on the grounds of the different constitution of the cerebrospinal fluid in various sections of the arachnoid sac or because substances introduced into the cerebrospinal fluid tended to spread by diffusion. As Jackson[55] stated, however, while there is no pump to maintain the circulation in the arachnoid sac, the fluid finds its way about much as does the venous blood from the extremities. There is the force exerted by the secretion of the choroid plexuses. This is materially assisted by gravity and by the pulsation of the brain. Tzanck and Renaut[54] have shown that the pressure in the dural sinuses tends to be lower than in the cerebrospinal fluid and that a circulation toward the dural sinuses is produced by a tendency toward a negative pressure. Weed and Hughson[55] arrived at the conclusion that the pressure of the cerebrospinal fluid under normal circumstances is from 5 to 50 mm. of water above the venous pressure in the saggital sinus. Frazier and Peet[72] proved that fluid made its way out through the aqueduct of sylvius by

placing a cannula in the aqueduct and noting the flow of fluid through the cannula. The fluid escapes from the fourth ventricle through the terminal opening of Magendie and the lateral clefts of Luschka to the subarachnoid spaces at the base of the brain beneath the tentorium cerebelli. Here are located the large cisterns of the pons, medulla and cerebellum. From these cisterns the fluid follows two main paths. The larger part rises over the surface of the midbrain through the narrow incisura tentorii to bathe the surfaces and sulci of the cerebrum and to receive accretions from its perivascular channels; it finally is mainly absorbed into the large venous sinuses. Another smaller portion descends along the ventral aspect of the spinal cord to the cauda equina and returns along its dorsal aspect to augment that stream which bathes the cerebrum. These two streams along the cord are only partially separated by the ligamenta denticulata and the spinal nerves between which they communicate the length of the cord.

THE ABSORPTION OF CEREBROSPINAL FLUID

Key and Retzius,[68] in 1875, during the injection of colored solutions under pressure into the subarachnoid space, were able to observe such solutions passing through the pacchionian bodies and into the superior longitudinal sinus. Dandy and Blackfan[73] were of the opinion that this work was subject to severe criticism because it was done postmortem and under high pressure. Katzenelbogen[61] stated that in view of the absence of pacchionian granulations in rabbits, in lower animals in general, and in infants, the hypothesis of Key and Retzius cannot be accepted. Weed,[74] in an experiment in which an isotonic solution of potassium ferrocyanide and iron ammonium citrate were injected into a subarachnoid space in which physiologic pressure was maintained, the animals killed and the brains immediately fixed in a solution containing hydrochloric acid, found that the Prussian blue granules were mainly in the arachnoid villi, chiefly along the superior longitudinal sinus and the cavernous sinuses. No granules could be detected in the ventricles, the choroid plexuses or in the perivascular spaces. He described an accessory or lymphatic pathway of absorption via the perineural spaces of the cranial nerves. Weed[71] stated that the pacchionian body was merely a hypertrophied arachnoid villus. It can therefore be seen that in consideration of the work of Weed the objections to the hypothesis of Key and Retzius are not valid, but that the arachnoid villus and pacchionian villus are essentially the same structure. Weed concluded that the main route of return of the cerebrospinal fluid into the general circulation is through the arachnoid villi; that a secondary portal of

exit is to be found in the lymphatic system; its participation in the absorption of cerebrospinal fluid is of importance, however, only in the isolated spinal subarachnoid spaces. He also thought that there was no evidence suggesting that the cerebrospinal fluid flows into the cerebral veins or capillaries, and that absorption of the cerebrospinal fluid from the cranial subarachnoid spaces proved to be faster and much greater in amount than the absorption from the spinal subarachnoid spacees. Kernohan[75] believes, however, that the arachnoid villus is of secondary rather than primary importance and assigns to the perineural "lymphatic" pathways the primary rôle. He comes to this conclusion because in elderly persons the arachnoid and pacchionian villi have practically disappeared, and because in MacLean's[76] recent study of thrombosis of the superior longitudinal sinus, in thirteen cases of a duration from five years to a few days, hydrocephalus was not identified in a single instance.

Wislocki and Putnam[77] attempted to answer the question whether or not substances introduced into the dilated and obstructed ventricle were absorbed, and if so, what is the pathway they follow from the ventricles into the brain. Their experiments were believed to demonstrate that a very moderate absorption takes place through the ependyma into the intercellular and perivascular spaces. Weed[78] demonstrated that the increase of the osmotic pressure of the blood following intravenous injections of hypertonic solutions induced aspiration of cerebrospinal fluid into the nerve tissue, the aspiration taking place along perivascular channels, but also through the ependymal lining of the ventricles.

THE DISTRIBUTION OF COMPONENTS BETWEEN THE BLOOD AND CEREBROSPINAL FLUID IN PHYSIOLOGIC AND PATHOLOGIC CONDITIONS

In summarizing the literature upon the concentration of diffusible blood constituents in the cerebrospinal fluid, Katzenelbogen[61] stated that they should be estimated not as absolute values but as compared with the concentrations of similar constituents in the blood plasma. Pathologic processes in the cerebrospinal nervous system may modify normal ratios of substances diffusible between the blood and cerebrospinal fluid in two ways: (a) by increasing or decreasing the barrier permeability from blood into cerebrospinal fluid and vice versa; (b) by modifying through metabolic process the content of diffusible blood components in the cerebrospinal fluid; (c) the ratios here considered may undergo changes even under normal conditions because of spontaneous fluctuations in the function of the barrier; (d) substances which originate partially or wholly in the cerebro-

spinal cavity should be evaluated as such and not as compared with similar substances in the blood.

CHEMICAL CONSTITUENTS OF CEREBROSPINAL FLUID

For the purpose of this paper, only the protein, chlorides, dextrose and pH of the cerebrospinal fluid will be considered, as these are the constituents of the spinal fluid variations in which have been interpreted as indicating specific disease entities. In this relation the statement of Fremont-Smith[79] and his co-workers are of particular significance. He said, "Even postmortem examination may fail to show the abnormalities responsible for variations in the composition of the fluids of the body. Rapid and significant changes may occur as the result of vomiting, diarrhea, food or fluid intake or transient changes in kidney function. It is our purpose to demonstrate the composition of the cerebrospinal fluid in relation to a given composition of plasma, rather than the composition of cerebrospinal fluid in relation to a given disease." In estimating the cerebrospinal fluid protein values in one hundred twenty-two patients who had been fasting nine hours or more before determinations were made, Fremont-Smith[79] and his co-workers found the values of non-protein nitrogen and protein to be as follows:

BLOOD SERUM

	Maximum	Minimum	Average
Non-protein nitrogen gm. per 100 cc.	42	12	27
Protein gm. per 100 cc.	8.2	6.1	7.0

CEREBROSPINAL FLUID

	Maximum	Minimum	Average
Non-protein nitrogen mg. per 100 cc.	28	12	19
Protein mg. per 100 cc.	43	12	28

This brings out the fact that the cerebrospinal fluid is an example of the intercellular fluid, a nearly protein-free filtrate of the plasma escaping through the capillary walls and bathing the tissue cells.

In reviewing the extensive literature on the dextrose in the cerebrospinal fluid, Katzenelbogen[61] summarizes as follows: The normal range of the cerebrospinal sugar values was found between 40 and 90 mgm. per 100 cc.; glycorrhachia below 40 mg. per 100 cc. should be considered as subnormal. The variations in sugar values found in the various stages of a disease such as septic meningitis makes the

value of these findings problematic. Cerebrospinal fluid sugar and blood sugar ratios have been found to fluctuate within wide limits in normal individuals (0.40 to 1.0) as well as in various diseases (0 to 1.01). The lowest figures were found in meningitis and brain abscess (0 to 0.27). The great lability of the cerebrospinal fluid sugar and blood sugar ratios in normals, in patients with various diseases and in the same individual under apparently similar conditions, renders attempts to utilize ratios for diagnostic purposes quite futile.

Katzenelbogen[61] concluded, after reviewing the literature, that in normal condition the pH shows nearly equal figures for blood and cerebrospinal fluid, namely, 7.4 to 7.6. In pathologic conditions changes in the blood pH influence the cerebrospinal fluid pH, but the latter generally remains more stable than the former. No specific diagnostic significance can be attributed to the pH of the cerebrospinal fluid either alone or in its relation to the blood. In reviewing the literature on acid base balance, Katzenelbogen[61] stated that normally the alkali reserve of the cerebrospinal fluid was equal to the alkali reserve of the blood, but that the alkali reserve of the cerebrospinal fluid was found to be lower than the alkali reserve of the blood in certain cases of meningitis.

In regard to the chlorides, Katzenelbogen[61] stated that in normal cerebrospinal fluid the chloride contents may range between 720 and 750 mgm. per 100 cc. Low contents of chlorides may be found in meningeal inflammation of any nature. In any case of meningitis, however, the cerebrospinal fluid chlorides may remain within normal limits at certain phases of the illness. Fremont-Smith and Dailey[80] found a definite relationship between the content of protein in the blood plasma and the concentration of chlorides in the cerebrospinal fluid. The higher concentration of chlorides in the cerebrospinal fluid than in the blood plasma compensates, according to Mestrezat,[81] for the higher protein content of the plasma in order that the normal osmotic tension between the blood and the cerebrospinal fluid may be maintained.

PATHOLOGY

Although we see many cases of disease of the middle ear and petromastoid or paranasal sinuses, only a few of them are associated with the syndrome of otitic hydrocephalus. This suggests that there may be some pathologic or congenital factor present in certain ones which predisposes to the development of otitic hydrocephalus. Since otitic hydrocephalus may be defined as an acute or subacute aseptic hypertensive intracranial state in relation to disease of the ear or

mastoid, any pathologic condition which may interrupt the pathways of the escape of the cerebrospinal fluid in otitic disease may be a cause of otitic hydrocephalus. A pre-existing pathologic state embarrassing the circulation of cerebrospinal fluid may be increased into a partial or completely occlusive state by a sudden edema or inflammation.

The lesions of syphilis and tuberculosis, together with tumors and cysts which might cause a latent hydrocephalus which could be actuated by an intercurrent disease may be properly left out of consideration here. Although recent writers on the subject, such as Cairns,[31] Symonds[1] and others state that little is known of the pathologic background of the symptom complex of otitic hydrocephalus, Quincke, in 1893[14] and 1896,[16] made some pertinent gross observations and was the first to suggest "serous meningitis" as a descriptive term for the clinical entity we are considering, and in his second contribution suggested an angioneurotic mechanism which was added to certain granular changes in the ependyma as a causative factor. Mya,[82] in 1900, grouped acute hydrocephalus, serous meningitis and ventricular meningitis and suggested acute ependymitis as a basis. In 1910, Merle[21] published his splendid thesis on "cerebral ependymitis" with a histologic study of lesions capable of producing a ventricular hypertension. His classification is extremely useful in separating the different clinical types.

In describing the gross appearance of cases of purulent and seropurulent ependymitis which Merle[21] states are approached and occasionally equalled in some cases of aseptic meningitis, he says that the ventricular walls appear blanched and the parietal surface is of a gelatinous consistence. The blood vessels appear obscured by a semi-transparent veil.

Histologically the epithelium under the influence of inflammation undergoes degenerative changes which lead to its destruction. Desquamation of the ependymal cells is frequent over a greater or lesser extent. Hypertrophic activity on the part of the subepithelial glial tissue may result in the formation of small mounds which protrude into the cavity of the ventricle. Merle concludes from his histologic studies of human material and material obtained from animal experiments that the ependymal epithelium does not constitute an effective barrier against infections in the ventricles, and he believed that the subependymal changes interfering as they did with capillary absorption were the most important. The subependymal cellular infiltration may be diffuse, but it is usually wholly perivascular. It is almost never lacking and appears quite clearly in

SYNDROMES OF INTRACRANIAL HYPERTENSION OF INFLAMMATORY ORIGIN (MERLE)

Acquired ventricular hypertension (bilateral)	diffuse inflammatory lesions	encephalitis leptomeningitis choroiditis ependymitis (usually the predominant lesion) meningo-ependymitis
	choroiditis	a very questionable lesion
	ependymitis	generalized lesion of the ventricular walls localized and obliterating the aqueduct of Sylvius and the fourth ventricle
Unilateral hypertension	phlebitis of the sinuses of the dura mater	
	ependymitis obliterating the foramen of Monro	
	adhesions of the ependyma isolating one part of the ventricular cavities	
Serous meningitis	edematous distension of the arachnoid meshes	generalized over the cortex localized: external pseudo-tumor

many cases where other inflammatory lesions are very slight. The perivascular spaces are distended by lymphocytes and mononuclear cells with large nuclei. The cells of the vessel wall become degenerated and mixed with the round cells and in many cases the vessels become partly destroyed. Thrombosis is frequent. The presence of polymorphonuclears in the perivascular space is rare. Merle[21] finds adhesions between the ventricular walls, especially in the regions of the horns, very frequent complications of acute ependymitis. Kernohan,[75] in a large number of autopsies done at the Mayo Clinic, has found this type of lesion present in but a single case. In regard to serous ependymitis, Merle[21] states that the lesions in these cases are often intermediate between the lesions found in acute purulent ependymitis and those found in chronic ependymitis. It is characterized by lesions, the inflammatory character of which is indicated by

diffuse cellular reactions and by masses of leucocytes surrounding the vessels. While there exist all intermediary stages between purulent ependymitis and these lesions, they equally tend to shade into a chronic ependymitis, and in particular, to the subependymal neuroglial sclerosis which is found in pachyependymitis. A weakness in Merle's[21] monograph lies in the fact that he was seeking to support the hypothesis that most of the absorption of cerebrospinal fluid was through the subependymal capillaries. Passot[2] called attention to the fact that in addition to the subependymal inflammatory changes accented by Merle,[21] edema of the ependyma and underlying brain tissue formed a prominent part in producing symptoms of acute and subacute intracranial hypertension.

Although attention had frequently been called to the effect of thrombosis of the cerebral sinuses in producing hydrocephalic states, the pathologic condition producing blockage of absorption from the pia-arachnoid sac without gross anatomic changes in the cerebral sinuses has only recently been explained by Weed[71] and Bateman.[83] Since the pacchionian body is, according to Weed,[71] merely an hypertrophied arachnoid villus, Bateman's[83] description of the pathology of the pacchionian villus may be taken to apply to both structures. He believed that the anatomicopathologic conditions of the pacchionian villi may be divided into two types; the acute and the chronic. In either condition the ultimate outcome is obstruction, either complete or partial. In acute inflammatory processes within the meninges the cellular exudate is most pronounced in and around these clusters of arachnoid granulations. Some of these processes advance to the degree of colliquation and produce complete obstruction directly or indirectly through rupture of the neck of the villus with the escape of fluid and exudate into the subdural space. Bateman's[83] work particularly indicates that there may be definite pathologic changes in the arachnoid and pacchionian villi, which might be sufficient to diminish the "filtration reserve" without producing clinical symptoms. Weed's[71] experiments indicate that those processes tending to block the pacchionian villi have an equal tendency to obstruct the accessory lymphatic pathways of drainage. The material presented by Merle[21] showing marked involvement of the subependymal capillaries, arterioles and venules present not only in acute inflammatory conditions, but also in "true hypertensive states," suggests that under certain conditions absorption into the subependymal capillaries might be important. It is unfortunate that Merle[21] did not include a study of the arachnoid villi and accessory lymphatic pathways in his thesis.

CAUSATION OF HYPERTENSIVE STATES OF PASSOT

In Quincke's[14] original paper on serous meningitis, he believed a simple serous effusion into the ventricles, such as is present in pleural and pericardial effusions, was sufficient to account for the syndrome of serous meningitis. In a later paper, 1896,[16] however, he suggested that an angioneurotic mechanism must be present. Curiously enough in 1937, Dandy[84] in his paper on intracranial pressure without brain tumor, apparently independently arrived at a similar conclusion. At the time of Quincke's paper, however, little was known about the physiology of the cerebrospinal fluid or the pathology of the meninges. Bateman[83] stated that from an anatomic point of view, any inflammatory process within the meninges may be considered obstructive, and that any form of increased intracranial pressure, including serous meningitis, can be considered as due to the same factor. He pointed out that while many authors attributed serous meningitis to oversecretion of cerebrospinal fluid by the choroid plexus, such a view is entirely in opposition to physiologic principles. Boschi,[85] in a comprehensive review of the literature, stated that in some cases of symptomatic hydrocephalus no abnormality has been found except edema of the subependymal tissue. He remarked that the presence of ventricular hypertension remains inexplicable in cases in which the choroid plexus is normal if one wishes to attribute the condition to pathologic changes in the plexus. The weight of the evidence, both as to the fact that the cerebrospinal fluid is a "biologic filtrate" and not a secretion, and the pathologic conditions found by Boschi,[85] Merle[21] and especially Bateman,[83] is in favor of hypertensive intracranial states being entirely obstructive phenomena except in the case of the rare papilloma of the choroid plexus (Grinker,[86] page 953). Gladstone and Dunlop,[87] however, showed that there was a vasomotor mechanism available for the brain, and suggest the analogy between intracranial hypertension and migraine first mentioned by Quincke.[16] Local obstructive vascular spasm and edema might therefore occur and produce the clinical phenomena mentioned by Quincke and Dandy.

In discussing communicating hydrocephalus, Grinker[86] states that subarachnoid hemorrhage during delivery may so block the arachnoid villi and perineural channels with fibrin and blood that acute hydrocephalus intervenes. He also states that hemorrhages of this nature may produce chronic changes in the arachnoid villi. D'Astros,[88] in 1898, thought that infection and inflammatory changes in the course of gestation were able to account for many cases of so-called congenital hydrocephalus. Politzer[89] pointed out

that hypertensive intracranial states may follow many acute febrile conditions in children, such as pneumonia, gastro-enteritis or scarlatina. Wallgren,[90] in 1925, was the first to suggest that benign lymphocytic meningitis was a definite clinical entity. This has since been thought to be due to a virus (Viets and Warren[91]), and Levaditi and Schoen[92] have shown that chronic proliferative and inflammatory changes in the nervous system of the monkey may be produced by the "virus lymphogranulomateuse" without provoking apparent clinical symptoms. Barker and Ford[93] have recently reported chronic changes in the spinal arachnoid space following benign lymphocytic meningitis. Dandy,[84] in his recent communication on intracranial pressure, makes special note of the small size of the ventricles in many of his cases. Merle[21] states that attenuated inflammatory lesions of the ependyma of an acute or subacute nature are rarely the only cerebral lesions which are present. One finds chronic lesions of the choroid plexus, of leptomeningitis or of diffuse encephalitis which have contributed to the realization of the anatomoclinical syndrome of acquired hydrocephalus.

It would seem probable, therefore, that one of the basic factors in otitic hydrocephalus in many cases, at least in adults, is some pre-existing preliminary pathologic change that seriously diminishes the reserve capacity of the foramina or of the arachnoid and pacchionian villi or of the perineural "lymphatic" spaces.

Passot[2] pointed out that acute hypertensive intracranial states were frequently seen in association with general toxemias, and that in otitis media or mastoiditis where there was no destructive osteitis or evidence of dural or meningeal inflammation, this symptom complex could only be produced by those ears which produced a general toxemia. Since Stern[67] has shown that toxins have the effect of lowering barrier resistance, the toxins might thus reach the ependyma, producing an inflammatory reaction sufficient to interrupt an already embarrassed circulation of cerebrospinal fluid either in the foramina or in the perineural channels and arachnoid villi. The hypothesis of a pre-existing pathologic condition would serve to explain the fact that hydrocephalus is not more frequently met with in toxemias in adult life. Stern[66] has also shown that the hemato-encephalic barrier functions poorly in the newborn, and this may serve to explain the appearance of this syndrome more frequently in infancy as reported by various authors.

While Frazer and Peet[72] found that the absorption of phenolphthalein from a ventricular system blocked by occluding the fourth

ventricle was almost nil, Wislocki and Putnam[77] demonstrated that in hydrocephalic animals a very moderate absorption takes place from the ventricles, the pathway being through the ependyma into the intercellular and perivascular spaces. It is therefore possible that under conditions of subclinical intracranial hypertension, absorption through the subependymal vessels might be a definite factor in absorption of cerebrospinal fluid. Merle[21] stated that several observations had demonstrated that compression of the vein of Galen and its obliteration could produce hydrocephalus, and he thought that it would be easy to understand that the perivascular infiltration and thrombosis of the vessels which he had demonstrated acting on the subependymal venous capillaries of the Galenical system could produce similar difficulties. Dandy and Blackfan[73] produced hydrocephalus in one of ten dogs by occluding the vein of Galen at its origin. Bedford[91] was unable to produce hydrocephalus in dogs by occluding the vein of Galen, and was unable to confirm the work of Dandy and Blackfan. He was inclined to agree with Cushing,[95] that the hydrocephalus was due to blocking the arachnoid villi with blood and the products of inflammation. The reports in the literature (Merle[21]) of cases of hydrocephalus produced by thrombosis of the vein of Galen suggest, however, that hydrocephalus can be produced by this means. If chronic pathologic change were present in the arachnoid villi or in the foramina, so that absorption adequate to prevent obvious clinical symptoms was just being maintained, inflammatory interference with drainage through the subependymal vessels or arachnoidal vessels in the spinal canal might be enough to precipitate an acute hydrocephalus.

Cushing[95] stated that any decrease in blood volume in the brain must be made up by an expansion of the brain or by an increase of cerebrospinal fluid, which probably amounts to the same thing. Weed[96] termed this a "thirsty brain" and demonstrated that the additional fluid which must necessarily be provided to compensate for the shrinking becomes sucked down into the perivascular channels. Cushing[95] further said that these changes all take place rapidly—a matter of minutes. "George Kellie was close to the facts a century ago when he said, in speaking of the effects of cerebral anemia, that watery effusion within the head is a pretty constant concomitant or consequence of great sanguinous depletion.' All this is part and parcel of the extraordinary phenomenon of cerebral edema, for the brain, owing to its sponge-like structure, is apparently more capable than any other of the body tissues of taking up fluids with an enormous and rapid increase in its bulk." It is therefore apparent that any condition which suddenly decreases the blood volume in the brain,

such as blood loss, shock, or other locally acting impulses from the autonomic nervous system, may cause a sudden edema of the brain which could precipitate a hydrocephalus in an already embarrassed cerebrospinal fluid absorption.

It is has been shown previously that the excess of chlorides in the cerebrospinal fluid balances the blood proteins as well as electrolytes. Weed and McKibbin[97, 98] and Weed and Hughson[55] have shown that on injection of hypotonic solutions intravenously a prolonged rise in the pressure of the cerebrospinal fluid was obtained with an outspoken swelling of the brain substance. The tendency to lowered chlorides which perspiration during and following operation produces with the low chloride diet of many seriously ill patients might again be a factor in precipitating a hydrocephalus. This calls attention again to the importance of supplying sufficient electrolytes in infections about the head. Kopetzky[99] has suggested that the lowering of the pH consequent on the increase of lactic acid in cerebrospinal fluid produces an edema of cerebral tissue which might interfere with the patency of the intraventricular foramina. If sufficient intracranial pressure were produced to interfere with the escape of blood via the cranial sinuses, the back pressure would immediately constitute a so-called "vicious circle" and tend to cause an ever increasing hydrocephalus.

It can be readily seen from a review of the physiology and pathology of otitic hydrocephalus that the theory of Ersner and Myers[38] has no validity, unless there is present sufficient pressure about the base to completely occlude the sigmoid and vertebral veins of both sides. This at once raises the question as to whether death following simultaneous ligation of both jugular veins may not be due to increased intracranial pressure, and whether the use of hypertonic solutions by bowel and vein and spinal puncture might not be of great value in such a case.

CAUSATION OF THE TRUE ASEPTIC MENINGITIS, SEROUS OR PURULENT, OF PASSOT

Passot[2] believed that the presence of increased proteins even without cellular elements, plus increased pressure in the cerebrospinal fluid, indicated a true meningitis which in the condition under consideration should be aseptic. In these cases he found marked destructive osteitis of the petromastoid with inflammatory changes of the dura, and frequently a latent suppurative labyrinthitis. In this condition he thought a direct local extension of toxins or bacteria to the arachnoid space from the endolymphatic sac was responsible.

That the presence of protein in the spinal fluid is not a completely valid differential point, however, has been shown by Stern[100] and her co-workers in their studies on the effect of toxins on the hemato-encephalic barrier. To establish the presence of an aseptic meningitis, Passot[72] insisted that (1) at least ten or twelve slides of the suspected cerebrospinal fluid should be examined under the movable stage with great care, having stained the slides with several different stains; (2) that several successive punctures should thus be found negative for bacteria; (3) that culturing should be done on several different media, according to the requirements of different organisms, and should be carried out under both aerobic and anaerobic conditions; (4) that inoculation into several different types of laboratory animals should give a negative result. He points out that even if all these examinations are negative, an organism might be localized by arachnoidal granulations or not possess sufficient vitality to grow on culture media, or be so few in number as to be missed on the direct smear, and suggests the term of "practically aseptic meningitis." He said, "This is not an idea conceivable to the bacteriologist, but is of great help to the surgeon in choosing the operative procedures which he should adopt." Jansen[101] also was of the opinion that "aseptic meningitis" was due to attenuated strains of organisms that failed to grow on the ordinary culture media. The reports in the literature of so-called serous meningitis, developing eventually into purulent meningitis with organisms, suggest strongly that Passot[72] is justified in this suggestion. The differential diagnosis between the simple hypertensive state and aseptic or sympathetic meningitis might be very difficult to make in an individual case of otitic disease. If the primary focus in the ear or labyrinth had been eliminated, however, the problem would be much simplified. The point stressed by Strauss[94] that sympathetic meningitis indicated a dangerous neighboring focus, justified Shambaugh[40] in considering aseptic and septic or infected meningitis in the same category, although obviously the difference in the gravity of the two conditions is considerable.

In all other respects but the foregoing the fundamental bases of the hypertensive states and serous or purulent aseptic meningitis are the same. That a serous or purulent meningitis may develop into a septic meningitis, that a hypertensive state may precede or follow a serous or purulent meningitis, or that either or both may eventuate in a chronic hydrocephalus can be readily apprehended. The attempt to differentiate between these conditions by laboratory tests alone is a futile attempt to substitute rigid formulæ for the experience and judgment of the surgeon.

SYMPTOMS AND SIGNS OF THE TRUE HYPERTENSIVE STATE (PASSOT)

While a didactic list of differential signs and symptoms is proper for the beginner, it is of inferior utility in actual clinical diagnosis, and so the erection of an edifice of differential points will not be attempted. A few signs and symptoms which may be present in hydrocephalus and conditions which might be confused with it will be mentioned.

Ocular Signs: The most prominent sign of the "hypertensive state" is papilledema, and visual difficulty may indeed be the only symptom present, especially if the hydrocephalus is of the communicating type. Merle[21] said that blindness is capable of occurring very rapidly and vision may be completely recovered, remain altered or be completely lost. Dupuy-Dutemps[102] has said that papilledema is a function of intracranial hypertention. The causes of papilledema are roughly: (1) increased intracranial tension; (2) optic neuritis; (3) obstruction at the optic foramen, and (4) pressure in the orbit sufficient to occlude the central vein of the retina. In a case of inflammation in the paranasal sinuses with intracranial symptoms, especially where there was a question of rupture into the orbit, or of an orbital cellulitis, it would be extremely difficult to decide from the presence of the papilledema alone whether an increased intracranial tension was present. The older writers frequently mentioned blindness as associated with intracranial hypertensive states, Hammond[103] and Oppenheim[18] particularly mentioning it as a possible sequel. Cairns[31] stated that arachnoiditis or serous meningitis simulates cerebellar abscess more than temporo-sphenoidal abscess. It comes on after acute mastoiditis and chronic mastoiditis, especially when the labyrinth has been infected. The main clinical feature is papilledema, often of extreme degree, and the headache and other general symptoms may be so slight that the condition is not recognized until sight is impaired. The case cited by Ramadier[15] in which papilledema was associated with increased ventricular hypertension is of interest, especially as Ramadier[15] suggests that these cases form a definite group and are due to an intracranial hypertension particularly involving the fourth ventricle. Hines,[104] however, assigns little value to this observation.

Photophobia is a rather frequently observed symptom. Oculomotor paralysis, both unilateral and bilateral, have been reported with ptosis and exophthalmos. Weakness of the abducens, usually bilateral, has been reported. The pupils may be dilated or contracted and loss of reaction to light has been observed.

Headache: Headache occasionally is an important symptom. It varies in intensity like most of the symptoms in the course of this syndrome. It may be diffuse, frontal or occipital. Cushing[105] has reported a case in which unilateral headache was present corresponding to the side of the dilated ventricle in which the foramen of Monro was obstructed by a granular ependymitis.

Vomiting: Vomiting is usually of the projectile type. Robert Whytt[3] stated, "The symptoms of no distemper resemble water on the brain so much as those which arise from worms in the stomach." Cushing[105] commented that, "It is not for us to smile at this," and went on to say that vomiting was one of the earliest and most persistent signs of hydrocephalus, "and whereas the vermifuge of Whytt's day might have given some relief by lessening the ventricular hydrops, the removal of the vermiform appendix, our modern tendency, would be far less likely to do so."

Nose: Anosmia has been reported by several observers.

Inner Ear: Nystagmus and vertigo are attributed by several observers to a preceding labyrinthitis, but in two of my personal cases[43] this symptom disappeared immediately following ventricular puncture, so that I am inclined to attribute it in those instances to increased endolymphatic pressure. In three unreported cases of otitic hydrocephalus I have observed a deafness with definite diminution in bone conduction but with a preservation of the higher tones and a loss of low tones present in both the involved and uninvolved ear. I concluded that this was in all probability due to stasis in the cochlea, tending to be most pronounced toward the helicotrema. I am not aware of this observation having been previously reported.

Signs of General Intracranial Pressure: The pulse may be rapid or slow, depending upon whether the irritative effect of the toxins or the slowing effect of pressure have the upper hand at the moment. Torpor and indifference have been frequently noticed. Eagleton[19] has described the semicoma which he ascribes to increased pressure in the pontile cisterns and deep coma has been noted by several authors. In cases where the left ventricle has been singly or predominantly dilated, aphasia has been noted. As Passot[2] stated, if the occlusion is confined to the aqueduct of sylvius, the retained fluid affecting the lateral ventricles produces symptoms of generalized cerebral compression which may be accompanied by symptoms of pressure on the Rolandic areas. When the foramina of Magendie or Luschka are closed, the hypertension involves the fourth ventricle and may produce symptoms of bulbar compression. Thus paralysis of the opposite side of the body, the same side of the face, and adiadokocinesis,

ataxia, rotation of the eyes to one or another side and other signs suggesting local disease of the temporal lobes and cerebellum have been produced. It is not to be forgotten that intracranial hypertension and brain abscess may coexist, as in a case I[106] reported in which meningitis sympathica and cerebellar abscess coexisted. Signs of meningeal irritation, such as rigidity of the neck and Kernig's sign, are not seen frequently in the hypertensive intracranial state. The reflexes may be exaggerated, diminished or abolished.

SYMPTOMS AND SIGNS OF TRUE ASEPTIC MENINGITIS (PASSOT)

In this variety of otitic hydrocephalus the signs of meningeal irritation such as photophobia, retraction of the neck, Kernig's sign and hyperactive reflexes are apt to be predominant, especially if the condition is acute and of the type sometimes referred to as sympathetic meningitis. If the condition is subacute, the symptoms and signs tend to approach those of the hypertensive state.

The leucocytes in the spinal fluid may be increased to as high a level as is seen in septic meningitis, occasionally being too numerous for accurate counting. The increased metabolism incident to the leucocytosis will result in lowered glycorrhachia, often as low as found in many cases of septic meningitis. The increased protein in the fluid will result in a lowering of the chlorides. Possibly metabolic factors may also result in a lowering of the chlorides. Increased glycolysis is apt to result in an increase in lactic acid. This substance, as well as other metabolities, are apt to produce a lowering of the pH of the cerebrospinal fluid. These latter factors, together with the interference with pathways of escape of cerebrospinal fluid already mentioned, may cause an initial aseptic meningitis to continue as a hypertensive state.

DIFFERENTIAL DIAGNOSIS BETWEEN OTITIC HYDROCEPHALUS AND CONDITIONS WHICH MIGHT BE CONFUSED WITH IT

The most important differential diagnoses are of meningitis with organisms or acute lymphocytic meningitis from the true aseptic meningitis, otherwise known as acute hydrocephalus, and brain abscess and nonsuppurative encephalitis from the true intracranial hypertension or subacute hydrocephalus.

Septic Meningitis: If there is a focus of infection in the ear or sinuses, there may be a continuous development from a serous to a septic meningitis. Laboratory procedures under these circumstances can give no more information than that the disease is in a certain stage of development. The practice of making a definite diagnosis of serous meningitis on the basis of laboratory findings and treating

the condition by spinal puncture alone can be a very dangerous procedure. This is evident from reports in the literature of cases diagnosed as serous meningitis which later developed into septic meningitis. The attempt to distinguish between serous and sympathetic meningitis upon the grounds of the absence or presence of protein or cellular elements in the spinal fluid, the one to be treated by spinal puncture alone, the other requiring elimination of the exciting focus, seems to me an impossible refinement of diagnosis in many cases. Such management, if attempted at all, would more safely rely on the experience and judgement of the physician than upon any laboratory tests whatever. Rather than to attempt differentiation with its consequent loss of time, complete removal of the exciting focus should be done in any case exhibiting symptoms of meningeal irritation. The most virulent forms of septic meningitis are associated with a cerebrospinal fluid almost if not entirely free from cellular elements. In these cases, however, the cerebrospinal fluid is swarming with bacteria and the diagnosis can be readly made by the pathologist. Treatment in such a case is futile.

Similar symptoms appearing after the primary focus of disease in the mastoid or sinuses had received adequate treatment would present an entirely different problem. While the problem of differentiating between true aseptic meningitis and latent labyrinthitis might occasionally present itself, the drainage of cerebrospinal fluid at frequent intervals should usually suffice, as was demonstrated by Hempstead's[107] cases of septic meningitis appearing after mastoidectomy which recovered with simple spinal drainage. If organisms could not be demonstrated in the fluid, this should be an entirely safe procedure.

Acute Lymphocytic Meningitis: Since Wallgren's[90] paper, in 1925, there has been a great and increasing interest in acute lymphocytic meningitis. Rivers and Scott[108] have identified a filterable virus as the etiologic agent. Recentely Viets and Warren[91] have reported a case with histologic examination of autopsy material. Nordwal[109] and Müller and Klan[110] have presented much material upon the differentiation between the virus type and those types due to other factors. Kuhn[111] has reported this syndrome following suboccipital puncture, Benard[112] and his co-workers after mercurial injection. Riesman[113] reported a case associated with tonsillitis and reviewed the literature on cases associated with acute upper respiratory tract infections. Due to its apparent frequency of occurrence, this condition might easily occur during an attack of otitis media or be confused with an upper respiratory infection. The differential

diagnosis may be easily made in the acute stage by the finding of a tremendous increase of mononuclear cells in the cerebrospinal fluid. An occasional case of acute lymphocytic meningitis may terminate in a hypertensive state and, if it were associated with an otitis, it would be quite impossible to differentiate the actual cause.

Tuberculous Meningitis: More difficulty is usually experienced in differentiating this form of meningitis from septic meningitis than from aseptic meningitis. However, otitis media and even mastoiditis on a tuberculous basis are not uncommon. Multiple perforations of the tympanic membrane with excessive granulation tissue in the middle ear are said to be characteristic of tuberculous otitis media. The finding of the tubercle bacillus on smear of the centrifuged pellicle is diagnostic, but this confirmation may be difficult to obtain. Guinea pig inoculation is a rather useless laboratory procedure, for the information gained comes too late. In my experience, the low cell counts found, combined with maximal symptoms of meningeal irritation when associated with a low glycorrhachia and relatively high chlorides, are very suggestive of tuberculous meningitis.

Nonsuppurative Encephalitis: Many authors since Oppenheim,[114] in 1900, have insisted upon the existence of a lesion of the brain which did not progress to brain abscess and gave minimal findings in the cerebrospinal fluid. Oppenheim[114] reported five cases. F. Voss,[115] in 1902, reported three cases associated with otitis media. Borries[22] and Yerger[27] each reported three cases in association with otitic disease, and Yerger[27] one in association with frontal sinusitis. In many of the reports the existence of an internal hydrocephalus has not been excluded. Ramadier,[45] however, reported a case in which one of the ventricles was tapped during an exploration for abscess without revealing either arachnoid or ventricular hypertension. That there is a lesion to correspond to this term seems possible, as especially the histologic findings of Borries[22] suggest, but on reviewing many of the case histories one suspects an insufficient diagnosis. These cases seem to show a strong tendency to recovery, are often impossible to differentiate from abscess of the brain or cerebellum and improve more rapidly after the dura has been incised. The differential diagnosis from otitic hydrocephalus has only been made by ventricular puncture.

Brain Abscess: The problem of differentiating from brain abscess the acute hypertensive state or stages of sympathetic meningitis in which the cell count is low is extremely difficult if not impossible in the individual case. Most signs show a tendency in one direction

or another, and while sometimes suggestive, are rarely diagnostic. The Ayala index test, according to Savitsky and Kessler,[116] has great differential value. A frequent differential sign between the hypertensive state and brain abscess is said to be the absence of leucocytes in the cerebrospinal fluid in the hypertensive state, but many cases of brain abscess have been recorded where leucocytis of the cerebrospinal fluid was absent, and cases of otitic hydrocephalus have been reported, as case 3 in my previous paper,[43] where the finding of a moderate number of polymorphonuclear leucocytes and lymphocytes was extremely suggestive of the presence of a brain abscess. Several cases reported in the literature suggest that a brain abscess may stimulate the production of an "aseptic" meningitis which may terminate in a hypertensive state such as the case of cerebellar abscess I[106] reported previously. An otitic hydrocephalus following in the train of a septic meningitis would be particularly difficult to differentiate, for as Caussé[117] stated, one must expect to see a brilliant operative success in septic meningitis transformed into disaster by the presence of an underlying brain abscess. It is hardly possible that any intradural inflammation could be uncomplicated by interference with the circulation or absorption of the cerebrospinal fluid in some degree, and it is therefore not strange that some difficulty is experienced in distinguishing between other types of intradural inflammation and acute hydrocephalus. The greater difficulty would seem to be in a case of brain abscess to decide which symptoms are produced by involvement of brain tissue and which by the almost inevitably present hydrocephalus. The matter has received a masterly summation by Nielsen and Courville,[35] who stated, "In view of the difficulties pointed out, how are we then to determine when in the course of an otitis media an abscess of the temporal lobe or cerebellum has developed and needs exploration (or whether a hydrocephalus is present)? The answer is not by one visit or even one careful examination, but by repeated systematic follow-up examinations plus a knowledge of what is apt to develop in such a case. To this is added diagnostic surgery which we do not hesitate to advise. When the ear drums have been opened and symptoms continue, we study the mastoids. When the mastoids have been drained, the dural and sinus plate removed and the lateral sinus studied, one has determined the presence or absence of extradural abscess and lateral sinus thrombosis. These procedures will, if carefully carried out, not only remove infection and hasten recovery, but will place the examiner in a position to know whether extension has occurred. This greatly simplifies the diagnosis. If the patient is known not to have extradural abscess, subdural abscess or meningitis, yet symptoms and signs of intracranial compli-

cations are still present, exploration for abscess of the brain is certainly justified." In other words, the differential diagnosis is best made by eliminating surgically in an orderly manner foci of infection about the dura which may tend to produce an inflammatory hydrocephalus, and when this has been done by exploration for brain abscess. If this exploration is negative, a ventricular puncture should be done. Those cases of the hypertensive state where the only symptom is loss of vision and the only sign a choked disk would not be difficult to diagnose if the possibility of otitic hydrocephalus is recalled.

Barré[118] has described a spinal arachnoiditis which may be independent or associated with a general arachnoiditis which he has divided into (1) the sensitive radicular form, (2) the radiculomedullary form, and (3) the radiculo-posterior horn form. The symptoms are those of irritation and paresis of the posterior roots and degenerations of the posterior horn cells. This condition, in association with otitic hydrocephalus, may account for some of the peripheral symptoms and signs pointing toward a brain abscess.

Diagnostic Tests: The tests used to distinguish septic meningitis from serous and sympathetic meningitis on the one hand, and hypertensive states from brain abscess on the other, depend upon cytologic and chemical examination of the spinal fluid, estimations of the pressure of the cerebrospinal fluid and upon roentgenographic examination of the arachnoid sac after injection of air. Widal's[119] test was the first exhaustive attempt to differentiate between septic and aseptic forms of meningitis by an estimation of "intact" and "injured" polymorphonuclear leucocytes. On examining the nucleus and the protoplasm, the following conclusions may be drawn: 1. The intact polymorphonuclear leucocyte is characterized by (a) a well outlined nucleus presenting a fine web of chromatin; (b) a granular protoplasm, slightly rounded. 2. The injured polymorphonuclear leucocyte is characterized by (a) a retracted nucleus in which the chromatic filaments have disappeared, which takes the basic stain strongly and uniformly; the structure recalls that of the normoblast. The nucleus is forced to the periphery of the cell; later it is fragmented into spherical balls. (b) Its homogenous protoplasm loses its granulations, presenting little clear vacuoles, round and unequal in size and arrangement; its contour is blurred and indented. According to Widal,[119] the first type of intact polymorphonuclear leucocyte proves the extravasation to be aseptic. The second type of injured polymorphonuclear proves the extravasation to be infected with pathogenic organisms. Passot[2] comments on this test to the

effect, that, like all biologic formulas, this law which will be correct in a great percentage of cases will not stand as an absolute rule for the individual case. From his own experience Passot has found this examination at fault (1) in fulminating disease where the evolution is too rapid for injury to the polymorphonuclear leucocytes to take place; (2) where the infectious agent is so attenuated that while it is able to call out a leucocytic response it is unable to produce any lytic action on the cells. In aseptic meningitis injured cells may be encountered when the polymorphonuclear leucocytes are called out by very virulent toxins or by less massive but prolonged intoxication.

Hempel[120] recently distinguished between aseptic meningitis and septic meningitis by a new method of examining living cells especially by infra-red light. He found that in the hypertensive syndrome the cell picture shows a rich polymorphonuclear leucocytosis with many cerebrospinal fluid lymphocytes, irritation forms, large mononuclears and degenerated ependymal cells. In the inflammatory case he found polymorphonuclear cells almost to the exclusion of any other type. Viets and Warren[91] found 98 to 100 per cent lymphocytes in their examination of the spinal fluid of a case of acute lymphocytic meningitis. Ayer and Fremont-Smith[121] presented a chart of average cerebrospinal fluid determination for many different conditions about the head. In addition to estimation of the cells, they give estimated values for protein, sugar chlorides and other chemical factors of less diagnostic value. They fail to give sufficient weight to the presence or absence of leucocytes in affecting the values for sugar and chlorides. Marked hypoglycorrhachia is usually to be found in acute infectious meningitis and not infrequently in the aseptic purulent type in which the intensity of the pleocytosis appears to be the determining factor. In any type of meningitis the content of the cerebrospinal fluid sugar seems to depend largely upon the phase of the disease, progression is usually accompanied by decrease, and improvement by increase of the original glycorrhachia. The chlorides in the cerebrospinal fluid, depending as they do upon the balancing of osmotic pressures between the blood and cerebrospinal fluid, will be profoundly influenced not only by the presence of cells and protein in the cerebrospinal fluid but also upon the variations of electrolytes in the blood. The variations in sugar and chloride values found in the various stages of a disease, such as septic meningitis, make the value of these findings problematic.

Ayala,[122] in 1923, produced the hypothesis that the residual pressure in the cerebrospinal fluid, after the extraction of a determined number of cubic centimeters of liquid, was related not only

to the initial pressure, but also to the quantity of liquid contained in the arachnoid sac. He stated that if one indicates the initial pressure by I, the final pressure by F, and the quantity of liquid extracted by Q, the formula $\frac{Q \times F}{I} = q^r$ will give a quotient (q^r) which is related not only to the initial pressure but also to the initial volume of the cerebrospinal fluid. The number q^r has been termed by Ayala[122] the "rachidial quotient." He has calculated this quotient in a number of brain tumors and cases of "serous chorio-ependymitis" and found that in the former the rachidial quotient varies between 2.55 and 4.55, while in the hypertensive intracranial state the quotient varies between 7 and 10. To secure a correct quotient, Ayala[122] advises that (1) the initial pressure be taken by a manometer before any fluid escapes; (2) that the final pressure be measured immediately after stopping the withdrawal of fluid; (3) that 10 cc. or more of cerebrospinal fluid be withdrawn and accurately measured. This test is also said to be useful in distinguishing between subacute hydrocephalus and brain abscess. Pollock and Boshes[123] deny that the rachidial quotient has any relation to the volume of the ventriculosubarachnoid system, but do not deny that this test may have an empirical clinical value. Savitsky and Kessler[115] studied 176 consecutive cases from the standpoint of the Ayala test. In all of ten cases of abscess of the brain an Ayala index below five was shown, and in six of seven cases, or 86 per cent, of otitic and sphenoid hydrocephalus an index of 5.5 was present. The index was found to be of greatest value in the differential diagnosis of these two conditions. In discussing this paper, Strauss[124] stated that consideration of the clinical picture was likewise important, and that one cannot rely on any technical method in making a diagnosis. He felt that when used properly, however, the Ayala index test was useful and a valuable addition to diagnostic technique.

While these tests are all of some weight in an individual case, in the present state of our knowledge they are always of distinctly secondary value to the experience and clinical judgment of the physician.

TREATMENT

Medical: In speaking of the practical application of Weed's[97] discovery of the effect of intravenous hypertonic fluid in decreasing the volume and pressure of the cerebrospinal fluid Cushing[95] said, "The surgeon promptly adopted the procedure as a means of lowering intracranial tension; and it is truly amazing how rapidly certain patients in deep stupor, even with the Cheyne-Stokes respiration, may be brought back to consciousness after the intravenous admin-

istration of a hypertonic solution. It was soon observed in my clinic that the effect of the concentrated solutions could be produced even when salt was introduced in the alimentary canal; furthermore, that the effects were more noticeable when the increased intracranial tension was due to hydrocephalus rather than to a solid tumor." It must be borne in mind that the introduction of hypertonic saline solutions intravenously must be considered an emergency measure, because just as the chlorides in the cerebrospinal fluid tend to diffuse into the blood, the chlorides tend to diffuse into the cerebrospinal fluid from the blood and an equilibrium will be reached eventually. Therefore, the effect of intravenous hypertonic saline solutions can only be temporary. The alimentary introduction of salt as Starling[63] demonstrated would produce a similar result somewhat more slowly. Some nonabsorbable hypertonic solution such as a tartrate or sulphate introduced into the bowel will have a less rapid but lasting effect.

The effect of limiting the fluid intake of the patient can be very marked in preventing the development and increase of intracranial hypertension. When a suspicion that interference with the absorption of cerebrospinal fluid exists, the fluid intake of the patient should be lowered to about 1500 cc. for the twenty-four hour period.

The effect of frequent small transfusions in preventing the development of intracranial complications has been stressed by Kopetzky.[99] He said that lactic acid which is normally present in the blood and spinal fluid in the blood-fluid ratio of 1 to 4 tends toward a marked increase in inflammatory states of the meninges. The presence of lactic acid in increased amounts must result in a changed iso-electric reaction of the spinal fluids and the cells bathed by it. Fisher[125] believed that lactic acid is the causative factor in the water-binding property of tissue colloids in the production of edema. Kopetzky[99] found that frequent small transfusions seemed to bring the pH toward the normal, increase the chlorides and the carbonates and markedly lessen the lactic acid content of the fluid. McGinty[126] reported that normal cerebral oxydation is accompanied by absorption of lactic acid from arterial blood. Conversely, when oxydation is impaired, excess production occurs in the brain cells with outward diffusion into the blood stream. Dickens and Greville[127] observed that respiration and glycolysis of slices of cat's brain was steadiest when, in addition to sodium, potassium and calcium were also present in the solution in which it was respiring: Potassium tended to increase respiration and glycolysis and the bivalent cations Ca and Mg lowered it. For example, if the Ca were left out of Ringer's solu-

tion, respiration was higher in the solution containing only Na and K than in full Ringer's solution. The action of Ca in keeping respiration low was not overcome by low concentrations of K, but it could be overcome by higher concentrations. They found it interesting that changes in the electrolyte content of solutions in which cerebral tissue is respiring exert such profound effects on its respiration but do not produce similar effects on other surviving normal or tumor tissue. They stated that ionic antagonism must also play some part in the mechanism by which calcium lowers respiration and potassium accelerates it. Similar antagonistic salt pairs are well known in biology.

It seems probable, therefore, that the effect of transfusion of blood on the electrolytes and lactic acid of the cerebrospinal fluid as suggested by Kopetzky[99] would be the effect of the injection of equal quantities of isotonic solutions containing the electrolytes of the blood. The effect of injecting small quantities of such solutions as Ringer's might be expected to have an equally favorable effect. Kopetzky and Fishberg[128] have also stated that the lowering of chlorides in the cerebrospinal fluid is dependent upon the increase in lactic acid and the lowered pH. It would seem more correct, however, to state that these findings were simultaneous than that one caused the other.

Surgical—Lumbar Puncture: While in a communicating hydrocephalus or an external hydrocephalus no harm can be done by lumbar puncture, Passot pointed out that the gravity of the symptoms may be increased in the noncommunicating type of internal hydrocephalus if lumbar drainage is performed. deMartel, Guillaume and Thurel[129] stated spinal puncture is contraindicated if signs suggesting involvement of the posterior cerebral fossa, such as lancinating occipital pain, loss of equilibrium and hypotonia are present. They point out that removal of the cerebrospinal fluid, if a block of the foramina or aqueduct or a brain abscess is present, may result in forcing the medulla into the occipital foramen. Symonds[1] was of the opinion that lumbar puncture with drainage of the excess of cerebrospinal fluid was the rational line of treatment in the hypertensive state. He pointed out that obstructive internal hydrocephalus was extremely rare. Merle[21] and Passot[2] both state that occasionally the drainage of cerebrospinal fluid must be repeated several times. This treatment is applicable to all cases of serous and sympathetic (aseptic) meningitis and especially in those cases of intracranial hypertension where papilledema alone or at most general signs of increased intracranial pressure are present. When localizing signs suggesting the possibility

of brain abscess are present, lumbar puncture should be done with extreme caution, usually only enough fluid being withdrawn to furnish a sufficient quantity for laboratory examination. It might be pointed out here, that if in the course of a lumbar puncture, signs of bulbar compression appear, an immediate ventricular puncture, done on both sides, if necessary, might be a life-saving measure. In any case where there is a suspicion of ventricular block it would seem wise to perform lumbar puncture in the operating room with facilities to do an emergency ventricular tap at hand. Weed, Wegefarth, Ayer and Felton[130] have suggested that withdrawing spinal fluid in the presence of a bacteremia tends to produce a septic meningitis by lowering the barrier permeability to bacteria. However, they were able to provoke septic meningitis in only six out of thirty-nine common laboratory animals, and clinical experience has not suggested that there is danger of producing a septic meningitis by spinal drainage. Ventricular puncture may be done to relieve symptoms as well as for diagnosis. When in a case where the presence of a brain abscess seems possible and either the symptoms are insufficiently localizing to warrant exploration of the brain or cerebellum, or the patient's condition is such that death seems imminent, ventricular puncture may be done as a preliminary measure to eliminate those symptoms due to intracranial hypertension. This may improve the patient's condition sufficiently to allow waiting for the optimum time for drainage of a suppurative encephalitis. Ventricular drainage is free from the danger of producing bulbar compression which is present with lumbar puncture both in brain abscess and obstructive ventricular hydrocephalus. The possibility that ventricular puncture might cause rupture of an abscess into a ventricle must be considered. It is questionable whether a rupture into a ventricle could be caused by this means, as a marked tendency toward a vacuum is not produced by removing the ventricular fluid, but the walls of the ventricles collapse together as the ventricular fluid is removed and the supporting force against them should not diminish. The factor producing rupture into a ventricle is encephalitis involving a ventricular wall rather than increasing pressure in an abscess. Ventricular puncture, by removing a factor tending to produce congestion which lowers the resistance of the brain substance, would tend to prevent rather than cause rupture. In regard to its effect upon a hydrocephalus, Warner[131] has reported a case of so-called idiopathic hydrocephalus in which the condition was successfully treated by repeated puncture of the lateral ventricle. He expressed the opinion that the value of removing as much cerebrospinal fluid as possible in such cases is attributable to the breaking of a vicious circle and the relief of

congestion of the choroid plexus. He would have been, no doubt, somewhat nearer the truth if he had placed the blockage of absorption in the ventricular walls due to edema and stasis or in the arachnoid villi. In possibly the earliest report on this procedure von Beck[132] observed the disappearance of coma and Cheyne-Stokes respiration and the return of vision in a few hours from the influence of ventricular puncture in a case of serous meningitis. On the other hand, when in an exploration for brain abscess associated with otitic disease nothing is found, the operation should not be terminated without exploration of the ventricular system. Unless a definite tract can be seen leading through the dura at the mastoid exploration, the logical procedure is to explore the brain through a clean field in order to avoid introducing infection. The ventricles should never be punctured through an infected field. It must not be forgotten that occasionally an abscess in the temporal lobe will produce cerebellar symptoms, so that in a case of suspected cerebellar abscess the temporal lobe should be explored if the cerebellar exploration is negative. The technique of choice in a suspected temporosphenoidal abscess appears to be that of Cushing, the subtemporal decompression, through which, if the exploration of the brain substance is negative, the dilatated ventricle is easily reached. As Passot pointed out, if the puncture of the first ventricle does not completely relieve the symptoms, the opposite ventricle should be punctured, as the fluid contained in the two lateral ventricles can be separated by arachnoiditis involving the foramina of Monro, and the puncture of one ventricle may not diminish the pressure on the other hemisphere.

Reports such as those of Strauss,[24] Shambaugh[10] and Williams[133, 106] illustrate what can be accomplished both in septic and aseptic meningitis by immediate and thorough surgical elimination of the exciting focus, and attention to drainage of the affected arachnoid sac when indicated. The point to be stressed is that failure to discover organisms in the spinal fluid should spur the surgeon on to the elimination of the exciting focus, rather than lead him to the dilatory inactivity of so-called "conservative treatment." It is true that occasionally in the hands of the otologist of great experience the expectant attitude may meet with success in an occasional case, but even under these circumstances avoidable tragedies occasionally occur.

Encephalography: Introduction of air into the subarachnoid space and ventricles by means of a lumbar puncture needle shows distortions of the ventricular system and to some extent of the whole arachnoid sac. Vincent[134] and his co-workers advise that the cerebrospinal fluid be completely removed and that toward the end of

the removal sufficient air be injected to keep the pressure in the arachnoid sac from falling too low. In this way headache and syncopal attacks may be obviated. Encephalography carries the danger of forcing the medulla into the foramen magnum, a possible fatal outcome for the patient, if there is any interference with the free passage of fluid from the ventricles to the subarachnoid space or from the subarachnoid space to the spinal canal in the presence of internal hydrocephalus. The presence of a brain abscess or cysts, particularly those in the region of the fourth ventricle, might also tend to produce this unfortunate termination. If there are signs pointing to involvement in the region of the base of the brain, or if there is a well grounded suspicion that a brain abscess is present, a ventriculography should be done.

Ventriculography: Vincent[135] and his co-workers advise that the ventricles of both sides be punctured simultaneously, as this not only aids markedly in the complete withdrawal of the ventricular fluid, but allows testing for the patency of the communication between the two sides. They also point out that following ventricular drainage, lumbar puncture may be done with a comparison of the chemistry and cytology of the two samples of cerebrospinal fluid. If the outline of the ventricles as shown by the roentgenogram is normal, the probability that a hypertensive state produces the symptoms of increased intracranial pressure is great. If an internal hydrocephalus is present, withdrawal of the trocar will be followed by a gush of cerebrospinal fluid, and if a communication exists between the several ventricles, all will be drained. Under these circumstances a normal encephalogram should be the rule.

Petit-Dutaille[136] has described at some length surgical measures for removing interference with drainage both of the ventricles and the subarachnoid space. These procedures, however, should be wholly in the province of the neurosurgeon and have but a minor interest for the otologist.

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LIV

PAPILLOMA OF THE TONSIL—WITH REPORT OF THREE CASES

IRA FRANK, M.D.

CHICAGO

True tumors of the tonsils, whether benign or malignant, are rare lesions, those of benign type being much less frequently observed than the malignant. Liveriero, in 1933, made a thorough study of the world literature (1827-1932) and found reports of only 150 cases of benign tumor of the tonsils. According to Vidau, the first case of benign tumor of the tonsil was reported by Robert in 1827; the first complete monograph on the subject was published by Ardenne in 1897.

The benign tumors which occur with the greatest frequency in the tonsil are the fibromas, papillomas, lymphomas, and angiomas; myomas, adenomas, lipomas, chondromas, teratomas and mixed tumors also occur but with far less frequency. Of Liveriero's 150 benign tonsillar tumors about half were fibromas, either pure or mixed.

In this report I wish to deal only with benign papillomas of the tonsil.

Pedunculated or sessile papillomas, or diffuse papillomatous hypertrophy of the tonsil, including epithelial elements, must be distinguished from the ordinary adenoidal lymphoid hypertrophy of the tonsil, although, from the clinical standpoint, the distinction is mostly of academic interest only, either condition calling for tonsillectomy.

In 1927, Vidau, who reported a case of hemorrhagic fibroma of the right tonsil, reviewed the subject of benign tonsillar tumors and states that in the world's literature he found reports of only 16 cases of tonsillar papilloma. Since then other cases have been reported by Haenisch, Landwehr, New and Childrey, Gandini, Tetu and Lazeanu, Ninomiya, Zoltán, Burger, and Markowicz. Haenisch reported one case which histologically was a true hard (keratinized) papilloma and showed evidence of inflammation. Landwehr reported five cases which histologically were proved to be hard papillomas. He does not state whether or not inflammation was present.

Fig. 1, Case 1. Note the lymphadenoid tissue (tonsil) and the tumor arising from the lining squamous cells. Hematoxylin-eosin preparation. x35.

New and Childrey reported the analysis of 357 cases of tumors of the tonsil and pharynx, observed in the Mayo Clinic, 1917 to 1930 inclusive; they found ten papillomas of the tonsil; they do not give a detailed description of these cases, except that the tumors were benign. Gandini, in 1932, reported nine cases of papilloma of the roof of the palate and tonsils; four of these were tonsillar. Zoltán, in 1935, reported four cases of diffuse papillomatous hypertrophy of the tonsils, all proved histologically. Burger, in the same year, reported three similar cases with histologic proof and Markowicz reported one hard pedunculated papilloma with histologic verification. Ninomiya reported two cases of tonsillar papillomatous hypertrophy and Tetu and Lazeanu reported the same condition, but I have not been able to consult the original reports.

In the records of the Department of Pathology of the Michael Reese Hospital, Chicago, for the past six years only three cases of hard papilloma of the tonsil came for examination. Short resumés of these three cases are given below. These cases are reported because of the rarity of reports of such lesions in the literature.

Fig. 2, Case 1. Hard papilloma of the tonsil. The same tumor as shown in Fig. 1. Hematoxylin-eosin preparation. x50.

REPORT OF CASES

CASE 1.—The patient, a white male, aged 28 years, presented a greyish-white, glistening pedunculated mass, measuring 1.2 cm. in all dimensions, at one pole of the tonsil. The clinical diagnosis was hard papilloma of the tonsil and tonsillectomy was performed. Histologic examination gave the picture of a squamous papilloma. The squamous epithelium extended in finger-like, sometimes branching, projections about a delicate core of connective tissue. The epithelial cells did not vary in morphology and the base of the projecting mass was well demarcated by the tonsillar epithelium. There were no signs of inflammation.

CASE 2.—The patient was a white male, aged 38 years, who showed a tumor of the tonsil, measuring 1.5 cm. by 1.5 cm., light grey in color and with a coarsely granulated surface. The clinical diagnosis was hard papilloma of the tonsil and the mass was removed. The histologic examination gave the same general findings as in Case 1.

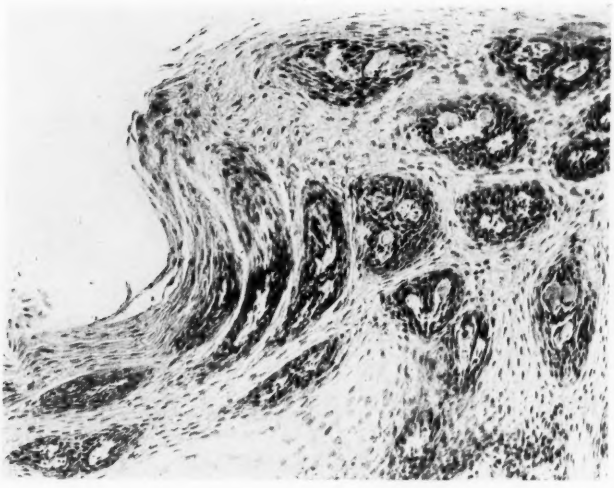

Fig. 3, Case 3. Hard papilloma. Note the horn pearls. Hematoxylin-eosin preparation. x125.

CASE 3.—The patient was a white male, aged 47 years, who showed a tonsillar tumor, pin-head in size, which was removed. Histologic findings were similar to those in Cases 1 and 2.

Clinically, papilloma of the tonsil is more frequently observed in adults than in children; it is, however, probable that in young subjects papillary hypertrophy may often be disguised under the general appearance of ordinary lymphoid tonsillar hypertrophy. Most of the cases have been observed in patients varying in age from eight to 40 years and there is nothing to show that sex has any influence. In the present report all three patients were males; in other reported series there was a preponderance of females.

Papillomas, like other epithelial growths, may be present as pedunculated, sessile or infiltrating growths (papillomatous carcinomas). They are generally unilateral, grey or greyish-white in color, and of multiform aspect. The tonsillar surface feels rough and granular, with verrucous excrescences, the tumor mass showing a cauliflower contour. If the tumor is pediculated it may protrude and is easily distinguished from a fibroma which is pale pinkish in color

and woody to the feel. If diffuse the warty excrescences may cover the whole tonsillar surface. Histologic examination gives the picture of papilloma with mucosal hypertrophy and squamous epithelial cells in connective tissue. Generally, the structure is that of a fibro-vascular core covered with stratified epithelium which may be keratinized. There may or may not be signs of inflammation. In the three cases now reported there was no evidence of inflammation; other authors such as Haenisch, report histologic evidence of inflammation.

The etiology of tonsillar papilloma is obscure and many theories have been put forward by different authors, based more or less on observations made in their particular cases. But none of the offered theories fit all the cases.

Cisler assumes that there are three factors concerned in the formation of hard papillomas: (1) the increased activity of the adenoid tissue in the young as a predisposing factor; (2) some direct cause of inflammation; (3) a mechanical factor due to accumulation of material in the lacunae. These assumptions stress inflammation. but, as previously remarked, not all cases show inflammation.

Gandini seems to favor irritation as a causal agent without however, stressing it.

Kleestadt's theory of hyperplasia on an infection-inflammation basis, seems to obtain most favor. It suggests that the upper pole of the tonsil is strictured or incarcerated and that development in a narrowed space, with stasis, favors a pedunculated growth formation. Ertl, Markowicz and others favor this theory. Haenisch also favors the inflammation basis of origin. However, the theory does not explain all cases. Inflammation is often absent as in the cases of Landwehr, Gandini and others. Hyperplasia is common to all these growths and would appear to be a result rather than a cause of them. Moreover, if infection be a basal factor papillomas should be observed much more frequently than they are as tonsillar infection and inflammation are very common. On the other hand, it is known that condylomata acuminata (which histologically resemble hard papillomas) are due to venereal infection and certain hard papillomas of the skin are said to be caused by a virus—Wile. These facts do not, however. prove the tonsillar papillomas are of infectious origin. Zoltán thinks that diffuse papillary hypertrophy of the tonsils may be caused by stasis, infection, or intoxication, among other factors.

Some light may, perhaps, be thrown on the possible formation of papillomas in the tonsils by investigation of the occurrence of such

growths elsewhere. Gehrmann showed that papilloma of the urinary bladder could be caused by long exposure to dyes consisting of nitro- and amino compounds. The possibility of the formation of papillomas in the urinary tract by chemical irritation is also referred to by Morson who, moreover, states that there is direct evidence that such growths may be produced by a parasite. Milian and Chapireau report a case of stomatitis with localization on the tonsil and in the subtonsillar cavity from the use of gray mercurial ointment.

A further useful observation is that of Lüscher of Berne who found that in hypertrophied tonsil cases the alkaline reserve is generally much decreased.

There is a possibility that papillomas, like certain other tumors, may be of embryologic origin. As pointed out by Hara, the union of epiblastic and hypoblastic elements in the tonsillar region makes it a very favorable site for the development of tumors. In the young, ordinary lymphoid hypertrophy may mask such tumors which are usually small in size.

It is thus seen that the question of the pathogenesis to tonsillar papilloma is still an open one and must remain so until definite proof of their origin is forthcoming.

As to whether or not benign tonsillar papilloma may become malignant is open to doubt. Some believe that benign tumors do not become malignant and that malignancy, if present, is a property which exists from the beginning. However, others, such as Despons, think that such tumors frequently undergo so-called malignant degeneration and this view is borne out by the known behavior of such growths in the urinary tract.

Little need be said in regard to symptoms, diagnosis and treatment. Subjective symptoms, in the case of diffuse papillomatous hypertrophy, are usually insignificant, perhaps some irritation and soreness of the throat. If the growth is pedunculated it may form an impediment to respiration and swallowing. In this case clinical diagnosis of benign papilloma can only be made following histologic examination of the tumor. Some cases have been diagnosed only after a tonsillectomy for ordinary hypertrophy. In the three cases now reported the clinical diagnosis of hard papilloma was verified in each case by the histologic examination.

The treatment is electro-coagulation or better complete removal of the affected tonsil.

SUMMARY

Three cases of hard papilloma of the tonsil are reported, verified by histologic examination of the removed tonsil.

The literature is reviewed and the clinical aspects, especially the etiology, are discussed.

104 South Michigan Ave.

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LV

THE LIMITATIONS OF BRONCHOSCOPY IN THE TREATMENT OF TRACHEOBRONCHIAL TUBERCULOSIS*

MERVIN C. MYERSON, M.D.

NEW YORK

The recent interest in tuberculosis of the trachea and bronchi has brought to the bronchoscopist greater opportunity for studying this subject. As a routine in many institutions for tuberculosis each patient is bronchoscoped shortly after admission. Bronchoscopy is gradually becoming a recognized procedure prior to the performance of thoracoplasty or other operations.

During the period between January 1st and May 3rd of this year, one hundred tuberculous patients were referred for bronchoscopy. Analysis of the bronchoscopies follows:

Ulcerations	4
Granulomata	9
Fibrotic lesions	6
Hyperplasia and subepithelial infiltration	7
Web across orifice of right upper lobe bronchus	1
Indentation of tracheal or bronchial wall	4
Areas of localized hyperemia	15
Carcinoma of bronchus	1

There were 31 definite tuberculous lesions of the trachea and bronchi.

The lesions which are encountered may be divided into two groups: The proliferative and destructive. The proliferative lesions include hyperplasia, tuberculoma, caseous infiltration and fibrosis. The destructive include ulceration and absorption of cartilages. All of the active lesions in the tracheobronchial tree have the ability to heal in most patients. For this reason local therapy is frequently valueless and at times harmful.

There is a large group of bronchial lesions in which there is no ulceration but a hyperplastic lesion which is the first stage of subepithelial infiltration. This hyperplastic process may exist with or without caseous necrosis and results either in ulceration or a fibrous stenosis. When the patient's resistance is good, instead of

*From the Department of Otolaryngology, Sea View Hospital.

caseation necrosis and ulceration, there ensues subepithelial and peribronchial fibrous tissue formation, which indicates a completely healed process. When the healing is incomplete one sees granulation tissue engrafted upon ulceration and recent fibrosis.

ULCERS

Ulcers begin as swollen nodules with central caseation, which break through the intact mucosa. The surface defect has an irregular margin which is thickened, and in some places undermined. The base is covered with necrotic material which, when wiped off, frequently reveals superficial granulation tissue. The granulation tissue is nature's attempt at healing. The deeper tissues may be uncovered by this process.

Ulcers may appear in the trachea or any of the bronchi. They are most frequently seen in the main bronchus, just beyond the carina and directly opposite the openings of the upper lobe bronchi. They are the result of interference with the circulation in the mucous membrane by the subepithelial infiltration. Ulceration is also caused by surface contact with the infected sputum. Subepithelial infiltration is found in the immediate vicinity of the ulcers and is without doubt the most frequent cause of their presence. If the subepithelial lesion does not resolve or become organized, an extension of the ulceration will result.

Treatment: Ulcers usually heal spontaneously. Those which do not heal spontaneously will not heal, no matter what is done to them. No matter what the treatment, the patient will not be benefited if he does not have the ability to form fibrous tissue. Active ulceration with a granular base and frayed edges, in the presence of active or spreading pulmonary disease, represents the type which is not replaced by fibrous tissue. These ulcers will be made worse by chemical or cautery application. They should be recognized and left alone. When, however, the ulcer is indolent and there is no evidence of acutely active disease in the immediate vicinity, superficial searing or stimulation with a mild chemical application is helpful. In this connection solutions of silver nitrate should not be used in greater than 5 per cent concentration. A solution of 10 per cent or stronger is destructive.

TUBERCULOMA

Tuberculomata are found chiefly in the lower trachea and main bronchi. They are occasionally seen in the introitus of a branch bronchus. We have seen such a granuloma on the inner edge of

a tracheotomy wound. Tuberculomata are very much like ordinary granulation tissue, except that they contain more mononuclear than polymorphonuclear cells. They are characterized by scattered epithelioid and giant cells and may contain tubercles. They have a tendency to undergo coagulation necrosis or caseation. Tuberculoma replaces the ulcerated mucosa of the trachea and bronchus. The superficial layers of the granuloma undergo caseation necrosis, shedding this caseous material in the form of an exudate which at times occludes the lumen. These structures are usually laden with bacilli and will account for a positive sputum in the presence of a well performed thoracoplasty or a well sustained pneumothorax. Tuberculomata very frequently heal and are replaced by fibrous tissue which always causes some degree of stenosis of the bronchus.

Treatment: The treatment of tuberculoma should be carried out only when the lesion is obstructive and causing symptoms. Because they are superimposed upon ulcerous lesions, and because they contain tubercle bacilli, manipulation of the tuberculomata may spread the infection to other parts of the lung. Therefore, biting forceps should be used only for superficial biopsy purposes, but not for the removal of the tumor mass. Unnecessary or prolonged manipulation of a tuberculoma should be discouraged. I have seen a case where prolonged manipulation with forceps caused a spread to the uninvolved lobes of the diseased side.

The cautery or coagulating electrode should be given preference when aiming to decrease the size of this lesion. Small, nonobstructive tuberculomata may be superficially seared or left alone, because they will disappear, unless the patient does not have the ability to heal. Where the patient is so handicapped, the tuberculoma will extend simultaneously with the ulceration under it. One or two cauterizations or applications of the coagulating current will suffice to stimulate circulation and cause absorption of its fluid content, thus decreasing its size. A large number of cauterizations does not seem to be indicated for this type of lesion.

FIBROTIC LESIONS

The largest group of obstructive lesions of the bronchus are classified as fibrotic. These constitute the intermediate and end results of all lesions of the trachea and bronchi. They result from ulcers, infiltration of the submucosa or of the peribronchial structures, or both; also from the healing of granulomata. The end result is a true cicatricial contracture of the involved tissues. The intermediate results follow upon the lesions enumerated and consist

Fig. 1, Case 1. Postmortem specimen viewed from behind, showing stenosis of the trachea and right main bronchus *A*. There is extensive thickening of the wall due to the tuberculous process.

of gradual fibrotic changes. The stenosis, when well established, is so firm that the strongest instrument cannot make an impression upon it. One of my associates broke a dilator while attempting to stretch such a fibrous constriction. These strictures are usually extensive. Therefore, attempted dilation is of no avail. It must be conceded that successful dilation of these stenoses is impossible. The presence of active disease in the bronchial wall favors its spread when a dilator is used. This occurred in Case 1. Intubation of the lower trachea by means of a special cane-shaped tracheotomy tube may be necessary to maintain the tracheal lumen in some cases. This will prove of only temporary value, as the stenosis will eventually shut of the lumen of the adjacent structure (See Figs. 1 and 2).

Subepithelial infiltration is a characteristic of tuberculous disease of the larynx, trachea and bronchi, and is seen in many sections of these structures, which in their gross appearance show no evidence of disease. Subepithelial infiltration can cause a complete and extensive stenosis of the bronchus. Extensive tubercle formation with lymphocytes, epithelioid and giant cells, with or without caseation, plays a prominent role in the production of stenosis.

Peribronchial infiltration occurs independently or as a part of the subepithelial process. It must be considered as an added factor in the production of stenosis. The combined form is the more frequent.

Infiltration of the bronchial wall by the tuberculous process may cause necrosis of the cartilages. Since these cartilages give form and support to the bronchial walls, their absorption causes a collapse of these structures. The bronchus then becomes widened and the lumen is replaced by a horizontal slit. The axis of the slit is always in a supero-inferior plane, while the compression is anteroposterior. This is because the cartilages occupy an anterior position.

Another form of bronchial lesion is the caseous endobronchitis. This is due to the accumulation of caseous material which comes from three different sources. It is frequently seen as a result of necrosis of the superficial layers of the granulation tissue which covers ulcerations. It is also found emptying into the main bronchus from the more peripheral structures; and lastly, it may be extruded through an opening in the tracheal or bronchial wall from a caseating lymph node. This material may be aspirated, but will usually refill the space it previously occupied.

Indentation and partial stenosis of the lower trachea and main bronchi are seen as a result of pressure by enlarged mediastinal

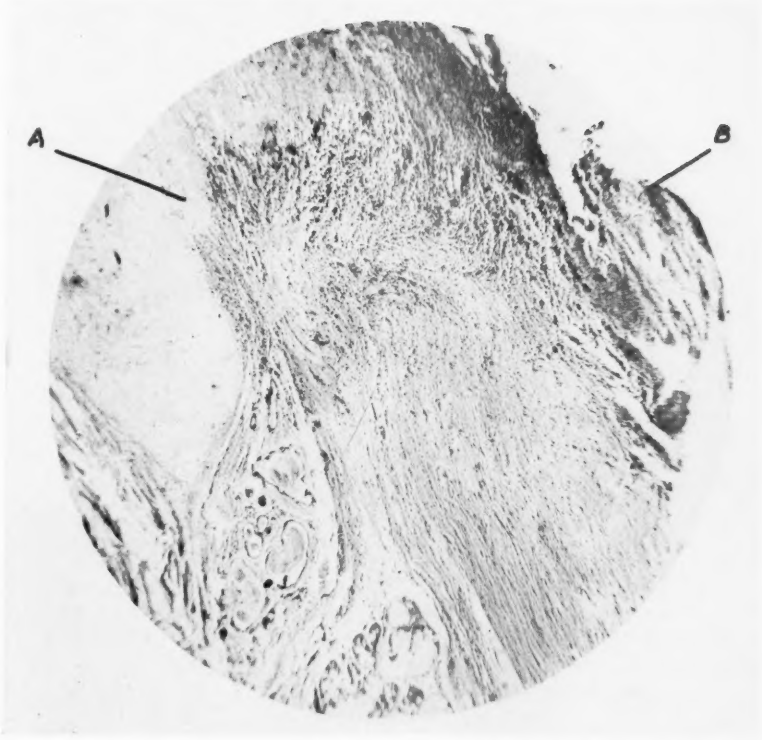

Fig. 2, Case 1. Photomicrograph of the wall of the right main bronchus illustrated in Fig. 1. Extensive caseation of the wall is seen invading and replacing the cartilage at *A*. Note also the protrusion of caseous material *B* into the lumen of the bronchus.

glands. We have not encountered a complete closure of the bronchus from such pressure, but have seen varying degrees of stenosis.

The following cases are cited to demonstrate that local therapeutic measures are not required for the healing of tuberculous lesions of the trachea and bronchi. They also indicate that there are limitations to the local therapy of these conditions.

REPORT OF CASES

CASE 1.—A white female, age 25, complained of cough and expectoration in April, 1933. A diagnosis of caseous pneumonic tuberculosis was made and right pneumothorax was instituted. While receiving the pneumothorax she had several attacks of severe dyspnea. Despite a good collapse of her lung, her sputum continued positive for tubercle bacilli. She was admitted in February, 1935.

At the time of admission a moderately loud wheeze could be heard. There were signs of a hydropneumothorax on the right side, while on the left numerous rales and rhonchi were heard. The roentgenogram confirmed the presence of the hydropneumothorax. The fluid aspirated from the pleural cavity contained tubercle bacilli in strong concentration. The physical signs and the persistent wheeze suggested the presense of an obstructive lesion in the bronchial tree.

Bronchoscopy performed by one of my associates March 6, 1936, revealed a narrowing of the lower trachea which was continuous with an almost complete occlusion of the right main bronchus. The left main bronchus was also narrowed. He attempted to dilate the stenosed right main bronchus. Following the attempted dilatation the patient developed a septic temperature; there was evidence of a massive spill to the entire left lung. Her condition became progressively worse and she expired the following September.

At autopsy the lower trachea, especially on its right side, was greatly narrowed. The lumen of the right main bronchus was reduced in size to about one millimeter, while the left main bronchial passage was reduced to approximately six millimeters in diameter. The right bronchial stricture occupied the entire length of the bronchus, while that of the left occupied a little more than one inch. Section of the right main bronchus revealed extensive caseous necrotic changes (See Figs. 1 and 2).

Comment: The autopsy specimen indicates the futility of attempting to dilate a stenosed bronchus. One cannot tell by looking through the bronchoscope the extent of the stricture. One should suspect the possibility of a caseous process in the bronchial wall when a stenosed bronchus is encountered. It should be borne in mind that there is great danger of spreading the disease in this type of lesion by instrumental manipulation.

CASE 2.—A white female, age 39, was admitted to the hospital October 17, 1934. At this time she complained of cough and expectoration, and some degree of dyspnea. Physical examination disclosed bronchovesicular breathing and coarse rales in the right upper chest. Diminished breath sounds and numerous sibilant and sonorous rales were encountered posteriorly in the left lower lobe. Tubercle bacilli were present in the sputum. The roentgenogram revealed diffuse productive

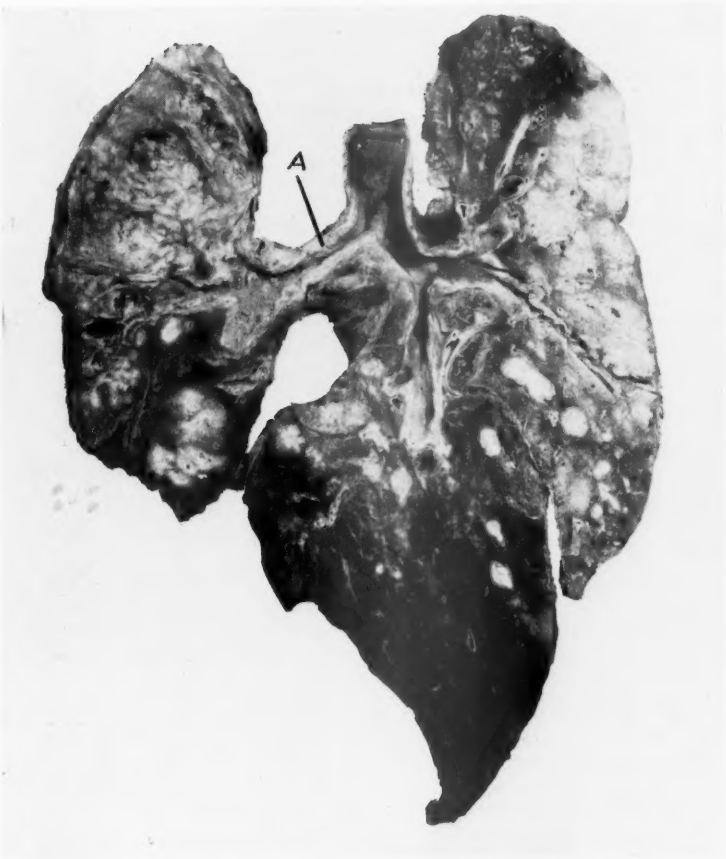

Fig. 3. Case 2. Postmortem specimen viewed from behind, showing extensive stenosis of the left main bronchus. Note *A* the marked thickening of the wall due to the tuberculous process.

changes throughout the left lung and the base of the right lung. There was slight retraction of the left chest and nodular lesions were noted on both sides. Obstruction of the left main bronchus was suspected.

Bronchoscopy revealed a greatly narrowed left main bronchial lumen, with a thickened mucous membrane throughout. A specimen was taken through the intact mucosa. Histologic study revealed tuberculous granulation tissue which was made up principally of lymphocytes, epithelioid and giant cells, and occasional tubercle bacilli.

Comment: Microscopic study of the thickened bronchial wall demonstrated the extensive subepithelial infiltration which exists in the walls of these stenosed bronchi (see Fig. 4).

CASE 3.—A white male, age 23, was admitted to the hospital January 26, 1937. He had had Pott's disease at the age of 3 and was perfectly well until 1933, when he complained of lower back pain. A spinal fusion was performed March 19th, 1936. Eight months later the sputum was found to contain tubercle bacilli, which disappeared after a Jacobeus operation was done. At the time of his admission there was a chronic lesion of the left upper lobe. The roentgenogram revealed a left pneumothorax; the left lung was diffusely opaque and was adherent in the region of the apex. The right lung appeared to be negative.

Bronchoscopy, June 23, 1937, revealed an ulceration on the lateral and posterior walls of the left main bronchus. This had a granular base. Thick, whitish, friable material appeared to be coming from the left upper lobe bronchus. Smears taken from the left main bronchus revealed the presence of tubercle bacilli. Biopsy of the tissue removed from the left main bronchus was recorded as tuberculous granulation tissue.

Second bronchoscopy, July 22, 1937, revealed a superficial ulceration of the left main bronchus, which was less in extent than the lesion previously noted. There was also a deep ulceration on the spur of the left upper lobe bronchus which was not seen before. Caseous material was seen coming from the left upper lobe bronchus.

Third bronchoscopy, Otober 28, 1937, there were no ulcerations to be seen. The upper lobe bronchial orifice was narrowed to a pinpoint. The left main bronchus was slightly narrowed.

Comment: This case demonstrates that ulcerations and granulomata will heal spontaneously. The lesions in the left bronchial tree were at no time treated.

CASE 4.—A colored female, age 26, developed a productive cough, fever, anorexia and loss of weight in November, 1933. Three years later she was transferred to this hospital from another institution. At this time the physical examination revealed evidence of a pneumothorax and a shifting dullness on the left. There were decreased breath sounds at the base. Fluid was aspirated and yielded large numbers of tubercle bacilli on smear. The sputum was positive for the same organisms. The Wassermann reaction was negative and the vital capacity was 1,300. On admission the roentgenogram disclosed a hydropneumothorax on the left with an uncollapsed cavity in the upper portion of the same side.

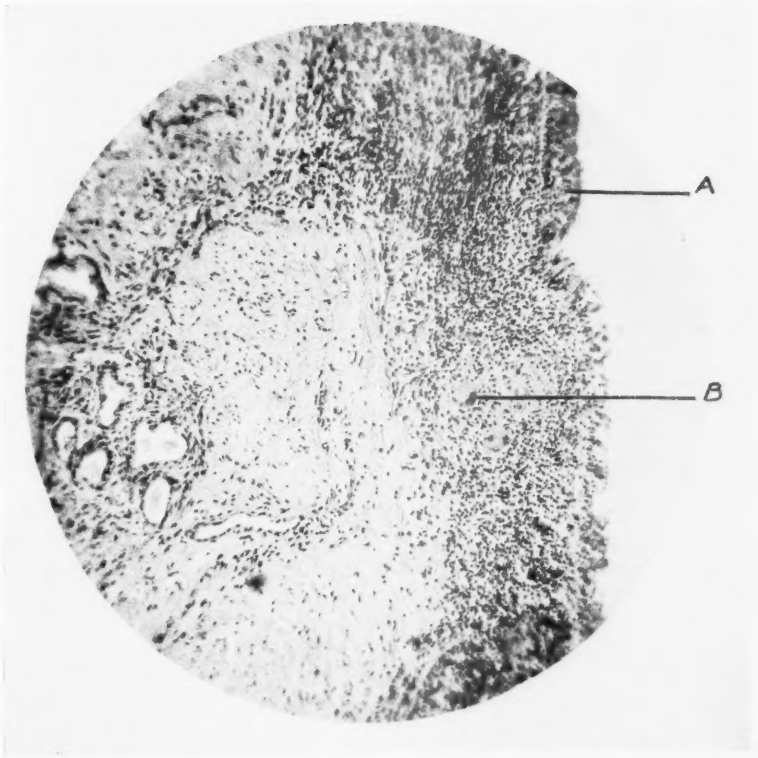

Fig. 4. Case 2. Photomicrograph illustrating non-ulcerative tuberculosis of the bronchus. Note *A* intact ciliated columnar epithelium, and *B* a typical epithelioid giant cell tubercle just beneath the epithelium. Such a process will be spread by dilatation.

Bronchoscopy, January 16, 1936, revealed an extensive granuloma occluding the left main bronchus. The histologic report was: Tuberculous granulation tissue.

Second bronchoscopy June 12, 1937, seventeen months later, revealed a long, tubular stenosis of the left main bronchus commencing about 2½ centimeters below the carina. The bronchus was narrowed to a lumen of about 6 millimeters in diameter. A small amount of thin, mucopurulent material was seen coming through this stricture with each expiration. The obstructing mass which was previously seen had disappeared; there was still some superficial granulation tissue occupying the walls of the strictured area.

Third bronchoscopy, January 12, 1938, seven months later, revealed a stenosis of the left main bronchus, which was now reduced to a lumen 4 millimeters in diameter. No granulation tissue was seen.

Comment: Bronchoscopic observation over a period of two years enabled us to witness the disappearance of a granuloma and its replacement by fibrous tissue. No therapy was used in this case. The original process disappeared, leaving the typical end result of a healed bronchial lesion, a fibrostenosis.

CASE 5.—A white female, age 32, was admitted May 1, 1935. She had pain in both sides of the chest in 1930 and hemoptysis the following year. Prior to admission she had been in several other institutions, where she had had pneumothorax therapy at various intervals. The physical and x-ray signs indicated disease of both lungs. There were numerous faint rales to be heard, especially on the right side posteriorly.

Bronchoscopy, August, 1935, revealed a superficial ulceration of the posterior tracheal wall, one-half inch above the carina, extending down to the region of the right upper lobe bronchial orifice. All other bronchi were free from disease.

Second bronchoscopy, April, 1936, revealed that the tracheal lesion previously noted was healed. The mesial wall of the right main bronchus now presented a superficial ulceration which extended down beyond the right middle lobe bronchial orifice.

Third bronchoscopy, June, 1936, revealed a small area of ulceration in the lower trachea posteriorly with scattered superficial small ulcerations in both main bronchi. The left main bronchus was markedly stenosed in the region above the lower lobe bronchial orifices.

Fourth bronchoscopy, July, 1937, revealed superficial ulceration and bleeding of the right main bronchus just beyond the carina. There were no other lesions to be seen except the previously noted stenosis of the left main bronchus.

Comment: The findings at bronchoscopy in this case, over a period of two years, indicate, on the one hand, nature's success in healing the ulcerative processes in the trachea and left bronchus, and on the other hand, the ability of the disease to reassert itself and appear in the right bronchus.

CASE 6.—A white female, age 53, was admitted to the hospital May 21, 1935. Her illness dated from 1931, when she complained of cough, expectoration and pain in the right chest. After a prolonged rest her sputum became negative for

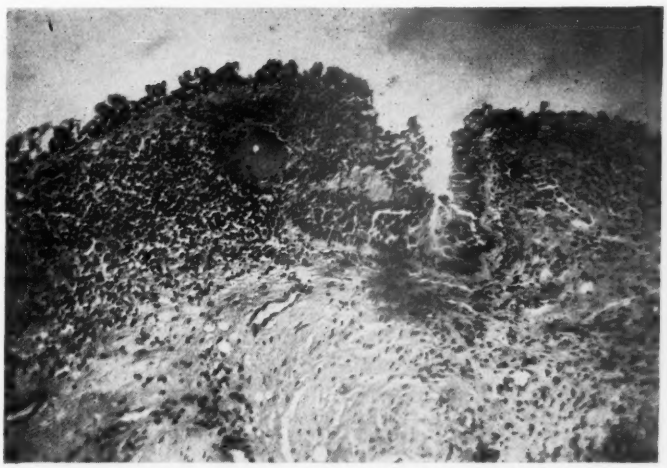

Fig. 5. Photomicrograph of bronchus showing an intact stratified columnar epithelium. In the submucosa there is extensive tuberculous granulation tissue in which a number of Langhan's giant cells are seen. The danger of dilating such tissue is obvious.

tubercle bacilli in 1934. At the time of admission bronchovesicular rales were heard throughout the right chest anteriorly, with exaggerated breath sounds throughout the left chest. There was dullness posteriorly on the right side. The larynx was negative. X-ray study revealed effusion in the right pleural cavity and a deviation to the right of the mediastinal structures. The last x-ray study made in January, 1938, revealed a diffuse opacity at the right base, evidence of atelectasis of the right lower lobe area and a marked deviation of the mediastinal structures to the right.

Bronchoscopy, May, 1935, revealed a granuloma obstructing the right main bronchus just beyond the opening to the upper lobe.

Second bronchoscopy, October, 1937, revealed a caseous necrotic lesion of the mesial wall of the right main bronchus extending from just below the carina to the middle lobe area. Here was seen a superficial granuloma engrafted upon an ulceration of the wall. The previously noted obstructing granuloma was no longer present.

Third bronchoscopy, November, 1937, performed preliminary to proposed surgery. At this time the bronchial picture was the same as a month before.

Fourth bronchoscopy, December, 1937, revealed that the superficial granulomatous lesion had extended farther toward the lower lobe branches. At this time an interarytenoid granuloma was found in the larynx.

Fifth bronchoscopy, March 7, 1938, revealed the anterior half of the carina greatly flattened and widened, with a superficial ulceration, in the center of which was seen a small white depression. This spot was thought to lead to a fistula which communicated with a caseating gland directly beneath the carina. The mesial wall of the right main bronchus was the seat of a superficial granulomatous process which reached into the branches of the lower lobe bronchus. This entire area was covered with a caseous exudate which could be aspirated.

Comment: This case demonstrates the progress of a caseous necrotic lesion of the bronchial wall over a period of almost three years. The nature and extent of the disease process was such that no form of therapy could have retarded its progress.

SUMMARY AND CONCLUSIONS

From the foregoing, it is apparent that bronchoscopy is of value principally as a diagnostic procedure in tuberculosis of the trachea and bronchi. The bronchoscopist is able to localize the cause of an atelectasis by finding an obstructing tuberculoma or fibrotic lesion. He can take smears from each bronchial orifice to ascertain the lobes from which the positive sputum is coming.

Except for the coagulation and cauterization of tuberculomata and the searing of inactive ulcers, there is little of positive value that the bronchoscopist can do in these cases. Silver nitrate should not be used in solutions stronger than 5 per cent; 10 per cent or stronger has a destructive effect.

Strictures cannot be dilated because of the type of cicatricial tissue which is present and because of their length. The average dilator is easily broken by the resistance of these firm fibrous stenoses. In addition, the possible presence of an active lesion in the subepithelial and peribronchial tissues makes dilation dangerous.

Aspiration is of value for the removal of secretions which are retained beyond obstructive lesions in the bronchi. Unfortunately, this procedure has only temporary value, since the secretions reform within a few hours and bronchoscopy at intervals frequent enough to keep the involved area clear is not practical.

Ultimate healing and recovery depend more upon the ability of tissues to react and heal than upon the therapy employed.

136 East 64th Street.

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LVI

OSTEOMA OF THE MAXILLARY SINUS*

A. G. Rawlins, M.D.

San Francisco

An osteoma is a more or less encapsulated bony tumor with unlimited growth displacing the adjacent structures, and usually attached to the bone from which it arises. While histopathologically it may resemble an exostosis, clinically there is considerable difference between them. Fetissof[1] states that an exostosis is a hyperplastic bony growth of a diffuse nature, usually on the basis of some inflammatory changes in the periosteum. In the case of an exostosis it is necessary to remove only the bony over-growth, while in the case of an osteoma it is necessary to remove the pedicle together with the bony tissue from which the pedicle is derived. Incomplete removal results in recurrence. Wiegmann[2] cites a case in which the growth recurred ten times following repeated incomplete operations.

The most logical classification of osteoma of the nasal accessory sinuses, as Hoover and Horrax[3] have suggested, is from the point of view of origin and not of the space they occupy. It is not always possible to tell the point of origin, however, and, as Bornhaupt[4] states, many of the so-called osteomata of the antrum of Highmore arise from the frontal and ethmoidal regions. Gerber[5] has classified these tumors according to the direction of their growth. Lalande[6] believes that this classification is based on secondary signs and leads to erroneous conclusions. Fetissoff says that it is illogical. It certainly does not give a correct physiological and anatomical foundation. In this thesis an osteoma of the maxillary sinus signifies an osteoma of and originating in the antrum of Highmore.

These tumors are histologically benign and do not invade the surrounding tissues by cellular extension, and they do not metastasize. They are malignant only in that they displace the surrounding tissues and erode them. In some of the older reports of very large bony growths (so-called "horned-men" and "frog-faces") it is very difficult to tell whether some of the cases were those of real osteomata, simple exostoses, luetic exostoses, leontiasis ossea or other systemic diseases. Before the days of antisepsis and asepsis, osteomata of the

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

antra were considered inoperable because the operative mortality was so high. Today these giant osteomata are rarely seen because surgical treatment is instituted before the tumors reach such huge proportions.

Reports show that osteomata are usually slow growing. Dahmann,[7] however, mentions one case where the growth was unusually rapid—only three weeks elapsed from the time the first symptoms developed until marked distortion of the orbit occurred. As Gerber[5] states, external trauma or infection of the sinuses sometimes stimulates growth. Suppuration and trauma may also produce a lack of blood supply with detachment of the pedicle. This produces a loose, dead osteoma free in the sinus cavity, as was reported by Hilton,[8] Stanley,[9] Duka,[10] Ledderhose[11] and Jaboulay.[12]

It is the concensus of opinion that this disease of the sinuses is rare, and it is certainly true that it is rare in the antrum. Only twenty-seven cases have been published up to the time of this report. Zuckerkandl[13] says that small osteomata are often found in the antrum at autopsy. Mieck's[14] thesis, in referring to Heyman's article, also mentions this fact. However, this could not be verified in Virchow's[15] Archives, where this statement was supposed to have appeared. Dr. A. W. Meyer,[67] Professor of Anatomy at Stanford University Medical School, states that he has not found a single case in about 1,000 anatomical specimens examined. In interviewing several of the roentgenologists of this vicinity, I find that only two such tumors have been seen in about 100,000 patients. Sjoberg[16] states that in twenty-five years the Otorhinolaryngological Clinic at Stockholm admitted 35,000 patients; of these 944 required radical antrum operations; 310 had malignant antrum tumors, and only two tumors were osteomata of the maxillary sinus. Hajek's[17] text contained no mention of this disease in the antrum.

HISTOGENESIS AND ETIOLOGY

The histogenesis and etiology of osteoma of the antrum are comparable with those in the other sinuses. There may be a difference in the ethmoidal tumors as this sinus develops from cartilage. Many theories have been reported on the development of osteomata. Among these are (1) development from the bony diploe (Virchow[15]); (2) development from the periosteum split (Bornhaupt[4]); (3) ossification of remnants of fetal cartilage (Arnold,[18] Boenninghaus,[19] Rokitansky[20]); (4) development from trauma and suppuration (Henrici[21]); (5) development from ossified polypus (Cloquet[22]); (6) development from osteophytes (Zuckerkandl[13] and

Dolbeau[23]). Dahmann[7] says that undoubtedly there are several methods of origin. Fetissoff believes that in the last analysis there are really only two theories: (1) development from embryonal cartilage, (2) development from embryonal periosteum. He states that the ossified polypus theory and development from bony diploe have only an historic interest. Guntzer,[24] Culbert,[25] and Gerber[5] give statistics showing that a large percentage of the osteomata arise near the time of puberty, and this fact favors some type of embryonic theory. Gerber[5] believes that irritants or trauma exert their maximum effect during the period of bone formation and account for the youthful age at which the tumors usually appear. A history of injury has been reported so often that it cannot be coincidental. As for the fetal cartilage theory, certainly an osteoma with its pedicle in the antrum, or one arising from the anterior and internal wall of the frontal sinus, cannot come from cartilaginous rests of the ethmoid. There are no histological points in favor of this theory. Also, as Fetissof says, we do not have to deal with osteochondroma. The case of Moure and Nadal[26] was the only one reported at that time. Halle[27] reported another case in 1930. Guntzer[24] states that none has been found with a trace of cartilage. A fair proportion have a co-existing sinus disease. Hucklenbroich's[28] statistics show that 37.5 per cent had a sinus infection. However, it has never been possible to determine whether the infection or the osteoma comes first. Pfeiffer,[29] who studied a young, growing osteoma carefully, believes that the tumor develops from the periosteum and that the bony growth results partly from osteoblastic activity and partly by direct metaplasia. Eckert's studies of a young osteoma revealed that the predominant cellular development determines whether the tumor is spongy, compact or mixed. His findings coincide with those of Pfeiffer.[29] He disagrees with Birch-Hirschfeld,[31] that the tumor is of endochondral origin, because the spongy bone network of the tumor is not just like the spongiosa of normal bone. Fetissof's work, which agrees with that of Pfeiffer[29] and Eckert, is so sound and convincing that his conclusions will be quoted. They are as follows:

"1. The growth of the osteoma takes place from the inside outward by the metaplasia of fibrous tissue into bone tissue.

2. The osteoblastic ossificatory process that takes place at the periphery of the osteoma plays a secondary role in the growth of the tumor.

3. The fibrous tissue filling the interstices of spongiose bone is the direct continuation of the periosteum covering the osteoma from the outside.

4. The theory of origin of osteomata from the periosteum, split off at the stage of the development of the organism, is the most fundamental."

He states that it is quite probable that an external irritant is also necessary, along with the periosteum split to cause the tumor. I believe that Fetissof in his histological studies of these tumors in different stages of development, has real proof for his conclusions. This method of development is the most likely.

ANATOMY AND HISTOLOGY

These tumors are hard, irregular masses, varying in size from a few grams to 440 grams. They grow expansively until the sinus wall may become thin and distorted. The tumor can even invade the overlying skin, the orbit or the intracranial structures. In recent years this has been uncommon.

The description of the cytology of these tumors is partly from the work of Eckert-Mobius, Fetissof, Garrettson[32] and several others, supplemented by the histological studies of two cases which I am reporting.

The microscopic picture is about the same as that of an osteoma of the other sinuses. The tumors are covered from the outside with ciliated, columnar epithelium overlying reticulated submucosa, interspersed with capillaries, round cells and an occasional mucous gland. Under the submucosa is the periosteum consisting usually of a thin outer and a thick inner germinative layer (especially in young tumors) which dips down into the mass of the tumor. The mass of the tumor may be one of three types: (1) ossea eburneum (hard or compact bone), (2) ossea spongiosa (soft, spongy or cancellous bone), (3) mixed (periphery compact and central portion spongy). In most cases the pedicle is spongy. The compact bone differs somewhat from normal bone, in that it is very irregular, with canaliculi extending in a parallel arrangement near the periphery and irregularly in bundles in the deeper parts. The compact bone may be surrounded by dense connective tissue passing directly into the bone. Only small areas are found with definite bony lamellæ arranged in concentric structure. In the spongy bone, which also differs somewhat from normal bone, there are irregular groups of interlaced trabeculæ surrounded by connective tissue growing into the bony structure. Fibrous tissue bundles make an irregular network throughout the tumor. In the central portion there may be polynuclear giant cells and large single nucleated cells. The picture varies somewhat, depending upon whether the tumor is young and

growing, or old and stationary. In the former case, fibroblasts may be seen in the bundles which, according to Fetissof, change into bone through metaplasia. At the stage of intensive growth there may be seen decalcification and ossification at the same time. Also, in these cases, near the surface the lamellar structure may be bordered by some osteoblasts. In the stationary tumors the connective tissue is usually very dense and there are practically no signs of activity.

SIGNS, SYMPTOMS AND DIAGNOSIS

From the smaller tumors there may be no symptoms whatever. Suppuration of the antrum is common. Neuralgic pain is present when the tumor presses on the infraorbital nerve. Epiphora is present when the pressure of the tumor causes obstruction to the tear duct. Exophthalmos often occurs and the eyeball may be pushed upward. Astigmatism and diplopia may be present when there is pressure on the eyeball (Howship's case[33]). There may be impaired vision or loss of vision from pressure on the optic nerve. There may be swelling of the cheek. The nasal wall is often pushed toward the septum, and the hard palate may be pushed downward. Cumiston[34] states that the anterior and lower walls of the antrum are more resistant than the upper and internal walls. The case of Howarth,[35] while not originating in the antrum, involved this sinus and produced a mucocele. This is indeed rare.

Antrum puncture may be of value in the diagnosis but it must be done with great care. Today diagnosis is made easier by the use of the x-ray. The slow growth of the tumor together with negative systemic findings and positive x-ray findings is usually indicative of osteoma.

DIFFERENTIAL DIAGNOSIS

The main diseases to be differentiated from osteomata are the malignancies, dental abscesses, osteomyelitis, cystic tumors, such as teratomata, fibromata, calcified fibromata and luetic exostoses. Lalande[6] reports a case of ossified fibroma and cites another mentioned by Virchow.[15] He states that there is a great similarity between an osteoma and a calcified fibroma. However, the calcified fibroma is not a growing tumor and is often calcified irregularly. Sometimes these two tumors can be differentiated only by microscopic examination.

Lalande[6] found many cases in the literature that have apparently been confused with osteoma of the antrum and do not properly come in this classification. This agrees with my findings. These cases are as follows:

In the case of Kammerer[36] the tumor invaded the antrum but originated in the ethmoid cells.

Paget[37] presented two cases of diffuse exostoses of the upper jaw.

The origin of Guntzer's[24] case could not be determined; it filled the whole nasal cavity and partly invaded the right antrum.

Coakley's[38] case was a tumor supposedly originating from the inferior turbinate. The patient had syphilis. There were several recurrences as well as other bony tumors which all improved under antiluetic treatment. Hence, these were probably luetic exostoses.

Morgan's[39] case, as well as that of Hutchinson,[39] was a large diffuse exostosis.

Davis'[40] tumor did not originate in the antrum but extended into it.

Del Pont's[41] case was diagnosed as an osteomyxoma.

Rodier's[42] patient had a calcified fibroma.

Windsor[43] admits that his case was probably an osteo-sarcoma.

Richet's[44] case was a crepitant, cystic tumor in the antrum. It contained some osseous trabeculæ which were filled with some gelatinous material. The diagnosis is in doubt.

Bruhn's[45] second case was probably a diffuse exostosis.

In the case of Lemere[46] the tumor filled the ethmoid, sphenoid, frontal and maxillary sinuses, but the origin was not known.

Halle's[27] case was an osteochondroma.

Fourdrignier[47] reported the removal of a bony tumor the size of a hazelnut from the region of the right antrum, but its origin could not be determined.

The discussion that followed Partsch's[48] case report places this tumor in the classification of leontiasis ossea.

Le Dentur's[49] case was some sort of congenital cyst in the antrum.

Bruhn[45] in his "Inaugural Dissertation" referred to cases described by Beatson, Ransford, Ferguson, Butlin[50] and Hancock as osteoma of the antrum. The cases that could be verified proved to be either malignant bone tumors or fibromata.

TREATMENT

The treatment of osteomata is definitely surgical. It may be done either by the Caldwell-Luc or the Denker method, depending upon the size and location of the tumor. Operation should be car-

ried out as soon as the diagnosis is made. Care should be taken to remove the pedicle and the bony tissue from which the pedicle arises.

Bornhaupt,[4] Phillip,[51] Boenninghaus,[19] Dahmann,[7] Eckert-Mobius[30] and Bajkay[52] have presented statistical studies indicating the rarity of antral osteomata. Sjoberg,[16] in 1935, after reporting two cases of his own, brought the number of osteomata of the antrum to twenty-one.

In my review of the subject, I have found twenty-seven cases; added to these are two cases which I here report, bringing the total to twenty-nine. These cases are as follows:

1. Howship[33] (1816). A male, age 40, whose maxillæ were distended, displacing the eyeballs. This was followed by blindness in both eyes and mental deterioration; finally death occurred at the age of 60. The specimen is in the Museum of the Royal College of Surgeons and shows osteomata the size of a fist in each orbit and originating in each antrum.

2. Cooper[53] (1818), states that he has in the collection at St. Thomas' a skull taken from a fishwife long remarkable for her hideous appearance. She had a large swelling under each orbit with projection of the eyes from their sockets. She died in an apoplectic attack. Examination of the head revealed a bony tumor growing from each antrum and projecting into the orbits and cheek bones.

3. Seutin[54] (1834). A male, age 34, who for two years had a bony tumor under the right lower eyelid. The eyeball was displaced upward and forward, and the right nasal channel was closed. The bony tumor was removed. It was covered with polypoid mucous tissue and apparently came from the antrum. The patient recovered.

4. Hilton[8] (1836). A male, age 13, who gradually developed a bony tumor under the left eye which distorted the orbit laterally. Through infection the tumor became loose, and when the patient was 23, the tumor extruded spontaneously, destroying the orbit and other structures, leaving a huge defect. The tumor weighed 440 grams and measured eleven inches in one circumference.

5. Michon[55] (1851), removed a large tumor from the right antrum of a man. It developed after an injury. The mass was covered with mucosa of the antrum and weighed 122 grams.

6. Paget[37] (1853), presented a report of a patient upon whom Stanley had operated, a man, age 37, who had a bony tumor of the right antrum. Suppuration and necrosis loosened the whole mass of bone with which the antrum had been filled. He was operated

upon and the whole mass pulled away. The appearance of the sequestrum, a hard mass of cancellous bone one inch in diameter, leaves no doubt of the nature of the disease. The patient recovered perfectly.

7. Paget presented another case also operated by Stanley; a girl, age 15, who had a bony tumor in one maxillary sinus. She was operated upon and died ten days later with erysipelas.

8. Schuh[56] (1862). A girl, age 14, with a swelling of the left cheek of two years' duration. The eyeball was pushed up slightly. There was no bulging of the hard palate. She had been injured by a blow five years before. An operation was successful in the removal of a bony tumor which, apparently, was an osteoma of the antrum. No microscopic examination was made.

9. Duka[10] (1865), had a patient with a large osteoma of the right antrum. The tumor was removed successfully with great difficulty and without the aid of an anesthetic. The tumor was lying loose in the antrum. It weighed 75 grams. Microscopically it showed nothing but irregular bone tissue.

10. Depres[57] (1875), reported a large so-called exostosis of the maxillary antrum which caused the eyeballs to protrude. Following operation, osteomyelitis and a cerebellar abscess developed, causing death. From the description, I believe that this was an osteoma.

11. Manz[58] (1879). A girl, age 18, who, two years following an injury to the left eye, developed a hard growth at the inferior angle of the orbit. At operation a large osteoma weighing 26.1 grams was found originating in the medial wall of the left antrum. The patient recovered.

12. Windmuller[59] (1890), in his thesis reported a case operated upon by Konig. A male, age 21, had noticed that the left cheek was gradually growing larger. The eyeball was pushed upward and slightly outward. Operation showed the antrum to be entirely filled with a hard, bony tumor. The patient remained well. No microscopic examination was made.

13. Bruhn[45] (1895), in Bergmann's Clinic, reported a boy, age 12, with a swelling in the right cheek and a right-sided headache. The superior maxilla was resected and a spongy, bony tumor was found. Microscopic examination showed it to be an osteoma.

14. Ledderhose[11] (1904), reported the case of a man, age 36, with a swelling of the right upper jaw which had been present since childhood. Suppuration occurred around one of the upper teeth.

At operation a free dead osteoma weighing 12 grams was found filling the antrum. The tumor had an ivory shell and a spongy interior.

15. Jaboulay[12] (1906). A man, age 25, at the age of 14 noticed a swelling in the right cheek. Infection of the right upper second premolar set in when he was 21, and intermittent pain followed. Curettage through the canine fossa was done, and eventually a radical antrum operation was performed. A free dead osteoma the size of a small egg and as hard as ivory was found. The patient recovered.

16. Phillip[51] (1911). A male, age 19, who complained of a left nasal obstruction of at least three years' duration. There was progressive asymmetry of the face. The left cheek bone appeared more prominent than the right. There was a slight depression of the left hard palate. He had no pain or exophthalmos. Anterior rhinoscopy showed that the left nasal fossa was filled by the sinuso-nasal wall which was pushed against the septum. Transillumination showed a cloudy left antrum. A Caldwell-Luc operation revealed an osteoma completely filling the left antrum. The patient recovered perfectly. Microscopic examination showed mostly fibrous tissue with a great number of osteoblasts in the midst of stratified osseous lamellæ, and osseous tissue in various stages of development.

17. Henrici[21] (1911), had a patient, age 18, who complained of lacrimation and impaired nasal breathing. Examination showed both nostrils obstructed from hard tumors which pushed the inferior turbinates and inferior meatuses against the septum on both sides. They appeared to be bony tumors originating in the antra, one of which was operated upon and part of the tumor was removed, with improvement in the breathing. No microscopic examination was done.

18. Rappe[60] (1924). A woman, age 64, had a tumor of the right superior maxilla for twelve years. The entire alveolar process was involved, but the patient suffered no discomfort. A diagnosis of osteoma was made by x-ray.

19. Leotta[61] (1919), had a patient with a hard swelling of the left cheek which finally involved the greater part of the left superior maxilla as well as the hard palate. A diagnosis of osteoma was made; x-ray findings agreed. A radical operation was successful. The microscopic examination proved the tumor to be osteoma.

20. Garnouec[62] (1927), reported the case of a woman, age 24, who had a tumor in the region of the right antrum which x-ray showed to be osteoma.

21. Garrettson[32] (1927). A man, age 46, complained of sinus trouble. The general examination and laboratory work were negative. There was pus in the right naris. X-ray examination showed dense clouding of the right antrum. Puncture of the right antrum was found to be impossible. A radical operation revealed an osteoma which was found originating in the floor of the antrum, growing upward and splitting the cavity. Microscopic examination showed fibrous tissue and cancellous bone.

22. Masuda[63] (1930). A girl, age 20, was found to have an osteoma filling the right antrum. Masuda was doing a bilateral radical antrum operation for suppuration of the sinuses. He had found no previous evidence of such a tumor.

23. Hertz[64] (1930). A woman, age 35, complained of pain in the right upper jaw of eighteen months' duration. A dentist had removed several teeth with no relief. A Wassermann test was negative. On operation the right antrum was found to be completely filled with osteoma.

24. Ruiz-Zorilla[65] (1932), on doing a radical antrum operation on a patient, age 21, for suppuration of the sinuses, discovered accidentally that the left antrum was entirely filled with osteoma. No x-ray pictures were taken.

25. Sjoberg[16] (1935). A woman, age 50, had a swelling about the right eye and right cheek. This was followed by suppuration and an infra-orbital fistula. The lateral wall of the right nasal cavity was dislocated medially and a polyp was present in the middle meatus on the right. Antrum puncture was attempted but found impossible. X-ray examination disclosed an osteoma the size of a tangerine filling the right antrum. An operation for its removal was successful. Microscopic examination showed a spongy type of bony tumor. The marrow spaces were filled with soft connective and fatty tissues. White, thin-walled vessels were found in the medullary spaces. In spots the tumor was covered with ciliated epithelium.

26. Sjoberg[16] (1935) (second case). A woman, age 32, whose left cheek had been swollen since childhood, had suffered severe pain in that region for some time. The floor of the right nasal cavity was dislocated upward. An antrum wash was done, but no pus was obtained. X-ray examination revealed an osteoma. A successful Denker operation was done, and a tumor was found arising from the antero-inferior angle of the antrum. The patient recovered rapidly,

Fig. 1

and she was discharged in ten days. Microscopic examination showed spongy bone. The bone marrow contained cells and a fibrillary substance resembling connective tissue.

27. Carmody[66] (1935). A male, age 11, four and a half years previously had developed a swelling in the left cheek. An operation was done in which a piece of bone was removed from the left maxilla. Later deformity of the teeth and an increase in the size of the alveolar process necessitated reoperation, and a bony growth which replaced the maxillary sinus was removed. Microscopic examination showed practically normal bone with no canals and only a few clefts.

I have two unpublished cases of osteomata of the antrum to report. One is my own and the other is one seen by Dr. Sterling Bunnell* of San Francisco. These cases are as follows:

*This case is published with the kind permission of Doctor Bunnell.

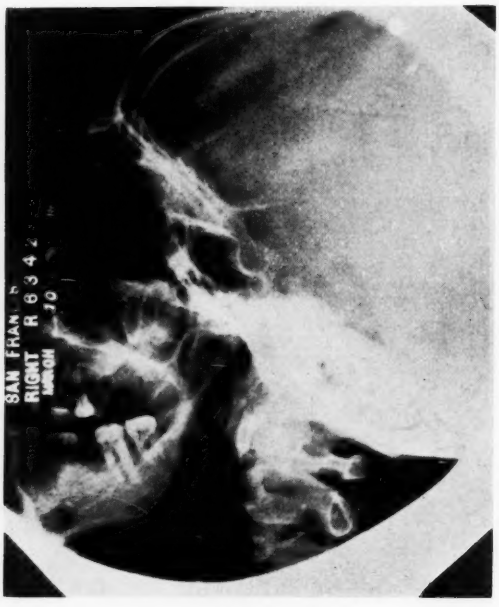

Fig. 2

REPORT OF CASES

CASE 1.—G. R., male, laborer, age 67, entered the San Francisco Hospital on the Stanford Service on May 2, 1933, with an acute mastoiditis on the left side. In the course of his general work-up, x-ray pictures (Figs. 1 and 2) were taken of his mastoids and sinuses. These films revealed a large, opaque mass of bone density in the right maxillary sinus. There was a slight amount of mucous membrane thickening in the left maxillary sinus; otherwise the sinuses were clear. After considerable deliberation an x-ray diagnosis of osteoma of the antrum was made.

The patient's history was negative, except that thirty-five years previously he had run a nail through the hard palate on the right side in the region of the alveolus. The wound drained for about two months and then healed entirely. He had suffered no pain or discomfort since that time. He had no headache and no swelling.

On examination there was pus in both middle meatuses. The only deformities present were a slight elevation of the floor of the nose on the right side and a small scar just back of the right alveolus. The rest of the physical examination and the results of the laboratory work, including Wassermann test, were negative.

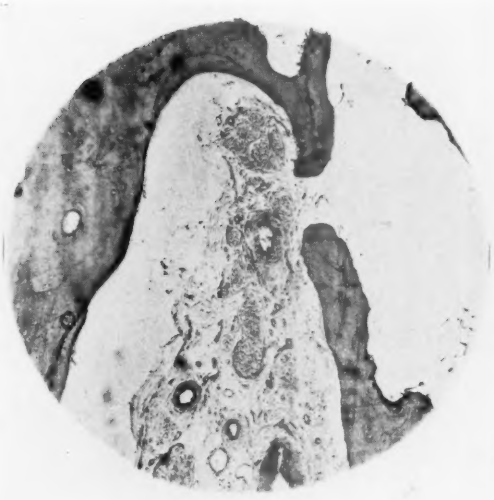

Fig. 3

One month after the patient had recovered from the mastoid infection, I operated through the canine fossa and found an osteoma almost completely filling the right antrum. Except for the posterior angle, the tumor was covered with mucous membrane and was attached by a pedicle at the anterior, inferior and mesial corner of the antrum. This was in the exact area the nail had penetrated. The tumor was removed by the Denker method. The postoperative recovery was normal, and the patient is well at the present time. In this case we have definite evidence that trauma is a factor in the etiology of some osteomata.

The pathological description is as follows: The specimen consists of three pieces of fine, spongy bone, the largest being 3¾ x 2¾ x 2 centimeters and having a thin, fairly smooth capsule which stripped easily.

Microscopic report (Dr. David Wood): "Sections (Fig. 3) show a tumor composed of cancellous bone. The bony spicules are of moderate width and rather compact in structure. The lacunæ are only slightly enlarged. The marrow spaces are filled with adipose connective tissue. No hematopoietic activity is seen. In one edge of the section, contained within a large marrow space, are a number of bundles of myelinated nerve fibrils (No reports of this finding in an osteoma could be found.) Nearby are several arterioles of varying width. The diagnosis is that of a well differentiated osteoma of the maxillary sinus."

CASE 2.—W. R., female, age 19, seen by Dr. Bunnell on February 4, 1937. There had been a painless swelling of the left side of the face for about twelve years, but more rapid growth had been noticed during the past two years. The

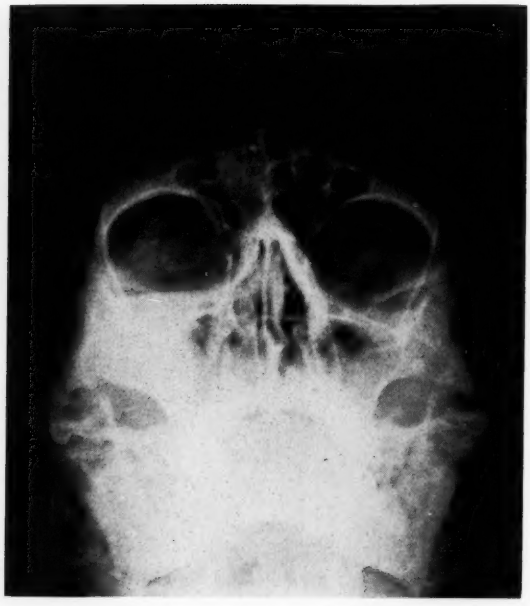

Fig. 4

swelling increased when she had a cold. There was no discharge from her nose except when she had a cold. She had no facial pain. Two years previously she had noticed that the left eye was displaced upward. The past history was negative except for the childhood diseases. There was no history of trauma.

The general examination was negative, as was the Wassermann test.

Local examination showed the left cheek to be bulging in the region of the malar bone. The left eyeball was pushed upward. The mouth and palate were normal in appearance. The eyes moved well in all directions. The vision was normal. X-ray pictures (see Fig. 4) showed a dense bony growth occupying most of the left antrum and apparently growing from its outer aspect and reaching from the palate to the orbit.

An operation under general anesthetic was performed by Doctor Bunnell on February 26, 1937. The left antrum was opened through the canine fossæ. After chiseling through the anterior wall, which was denser than normal, he encountered spongy bone occupying the whole antral area. This bone was curetted out, leaving a sort of hard shell which was then about one and a half inches in diameter. The posterior inner wall of the tumor was then removed with a chisel, entering the antral cavity. The partition between the two cavities was chiseled away, and a large opening made in the inferior meatus. Convalescence was painless and prompt.

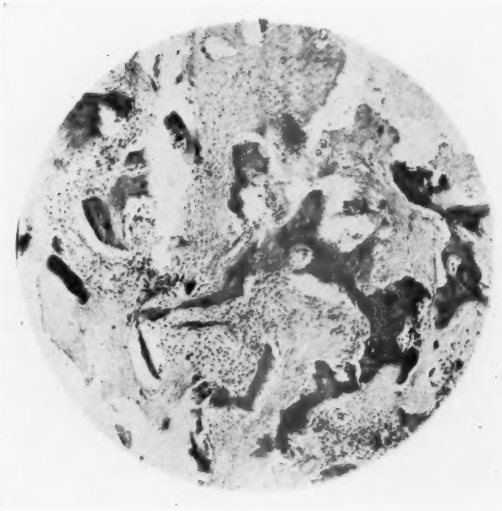

Fig. 5

Pathological report by Dr. David Wood: "The gross specimen consists of many small fragments of bone and soft red tissue.

Microscopic report (Fig. 5): Sections show no mucous membrane and no structure which can be definitely interpreted as periosteum. (The muco-periosteum from the first tumor was removed and lost. The second tumor was removed piecemeal and hence had no capsule.) The specimen is extremely fragmented and consists entirely of osseous bone, the marrow spaces of which are filled with cellular fibrous tissue."

The first osteoma described was well differentiated and adult in type. This is in contrast to the one just described. In the former tumor the bony spicules are well formed, uniform in shape and size, well calcified, and are associated with an adult type of fatty marrow. No osteoclastic or osteoblastic activity is seen. The myelinated nerve described above was near fairly large arterioles and in all probability was following a nutrient artery into the tumor.

The other tumor was of the young type. Its spicules varied considerably in uniformity of size, shape and calcification. Occasional osteoclasts are seen. Moderate osteoblastic activity is present. Another sign of the youthful nature was the cellular fibrous tissue

contained within the marrow spaces. This varied considerably in amount and cellularity, although no areas of hyalinization were found. It is quite possible that the young connective tissue shown in Fig. 5 could, through further development and metaplasia, change into adult bone, as shown in Fig. 3.

SUMMARY

Thirteen patients were male, eleven were female, and in five cases the sex was not stated. This does not give any great preponderance for the male sex as has been stated in most of the literature. Three cases were reported as being bilateral. (Birch-Hirschfeld in 1920 had found two such cases); fourteen were on the right side; eight were on the left side, and the side was not stated in four cases. Twenty-nine years was the average age. This definitely agrees with the statistics of all the authors. Taking into consideration the fact that these tumors often grow for many years before symptoms arise, even the few older patients reported may come under the age of development of the facial bones. Two deaths followed operation, and two nonoperated patients died from extension of the tumor. Of the twenty-nine cases, twenty-three were operated upon and six were not. This makes a mortality rate of 8.7 per cent in the operated cases and 40 per cent in the nonoperated. There was a history of injury in 20 per cent of the cases, and a history of associated sinus infection in 28 per cent. There were five dead osteomata in the total of twenty-nine.

CONCLUSIONS

1. Osteoma of the maxillary sinus as defined and classified is a very rare tumor. The total number of cases to date is twenty-nine.

2. The theory of development suggested by Fetissof is the most convincing. He concurs with Pfeiffer and Eckert-Möbius in his histological studies.

3. Trauma is a factor in some cases. This is definitely proved by my first case report.

4. Associated sinus disease was not present in enough cases to justify positive conclusions.

5. Osteomata usually develop during the growth period of the facial bones.

6. No preponderance in the male sex was found.

7. Roentgenograms simplify diagnosis in most cases. It is difficult only in the rare condition of calcified fibromata.

8. The treatment is always surgical. The pedicle and surrounding bone should be removed.

9. Modern surgical technique has made the mortality rate negligible. The last death reported following operation was the case of Depres in 1875.

384 POST STREET.

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LVII

HIATAL HERNIA*

HERMAN J. MOERSCH, M.D.

ROCHESTER, MINN.

Diaphragmatic hernia has been a source of interest to the medical profession for many years. The earlier studies dealing with this problem dealt primarily with hernia occurring as a result of trauma, or hernia of congenital origin developing through the anatomic points of weakness existing in the diaphragm. With the improvement in roentgenographic technique, it became increasingly apparent that the most frequent site for diaphragmatic herniation was through the esophageal hiatus. The herniation of part or all of the stomach through the esophageal hiatus may give rise to many bizarre clinical manifestations. Increasing experience has taught that esophagoscopy may be of the utmost importance in a proper evaluation of such symptoms and it may be an important factor in the differential diagnosis of the type of hernia and a guide in treatment.

Diaphragmatic hernias may be divided into two types, the true and the false. The true hernia is characterized by a peritoneal sac separating the hernial contents of the abdominal cavity from the thoracic or mediastinal space. The false hernia possesses no such covering and a direct communication exists between the peritoneal and thoracic cavities. Esophageal hiatal diaphragmatic hernias, almost without exception, are of the true type.

Esophageal hiatal diaphragmatic hernias, or hiatal hernias, which term will be used for the sake of brevity, have been divided by Åkerlund[4] into three types which are especially of importance from a therapeutic standpoint: (1) Congenital short esophagus with partial or complete thoracic stomach (Fig. 1); (2) esophageal hiatal hernia without short esophagus and in which the esophagus does not form a part of the hernia; that is, para-esophageal hernia (Fig. 2); and (3) esophageal hiatal hernia in which the esophagus is shortened but

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Division of Medicine, The Mayo Clinic.

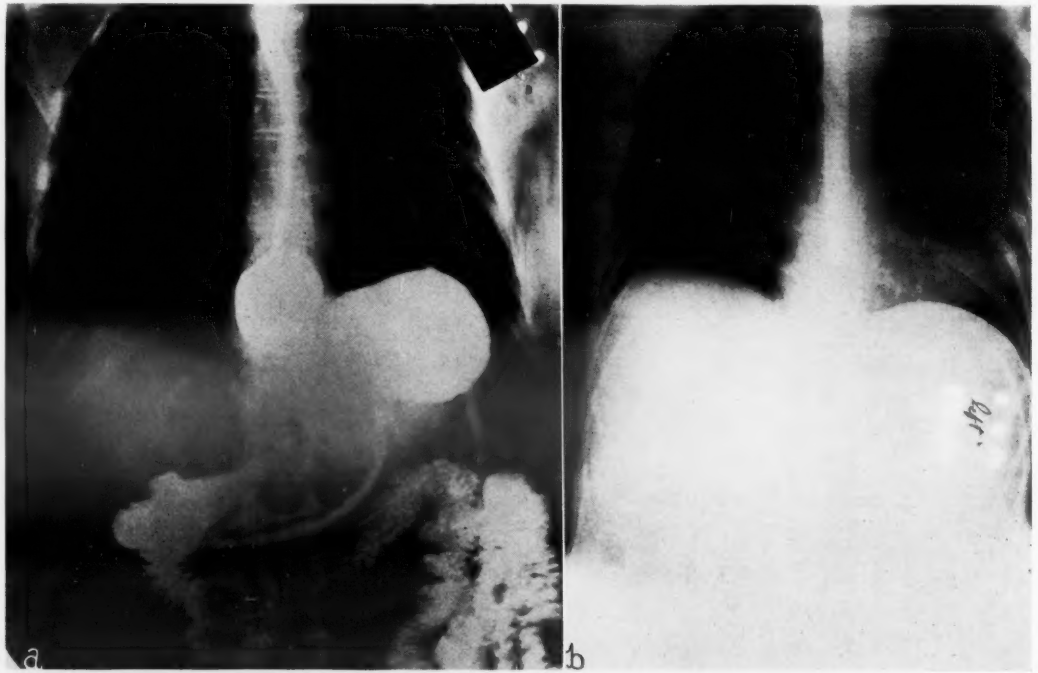

Fig. 1. *a*, Congenital short esophagus with partial intrathoracic stomach. *b*, End of esophagoscope at level of esophagogastric junction. Tissue removed at this point was reported as gastric mucosa.

in which the distal portion of the esophagus is a part of the hernial contents (Fig. 3).

It is not possible within the confines of this paper to enter into a detailed discussion regarding the etiology of the various types of hiatal hernia. Suffice it to say that Bailey,[1] Bund,[2] Findlay and Kelly[3] and others have demonstrated that congenital short esophagus with intrathoracic stomach is invariably owing to embryologic retardation. The possibility of such a picture occurring as a result of cicatrization of the esophagus cannot be dismissed (Fig. 4). The etiology of the two other types of hiatal hernia is not as obvious. It is generally regarded, however, that they are of congenital origin in the same sense as inguinal hernia, and any factor that tends to increase intra-abdominal pressure may act as a contributory agent.

The present study of hiatal hernia is based on a series of 267 cases of diaphragmatic hernia encountered at the Mayo Clinic during the period of five years from 1932 to 1937. Although a similar diagnosis was made in other cases, only those cases are included in which there was positive roentgenographic evidence of such a lesion. The most striking fact was that 246 of the 267 cases were of the hiatal type of hernia, in contrast with fifteen traumatic and six congenital diaphragmatic hernias other than the hiatal type.

My experience is in accord with that of Akerlund[4] and that of Ude and Rigler,[5] that the third, or sliding type of hiatal hernia is by far the most frequent, and the para-esophageal type, the least frequent. No attempt was made to classify the hernias into one of the three types, because we have found, as will be pointed out later, that it is sometimes impossible to distinguish accurately one type of hernia from another (Fig. 5).

The distribution of the 246 cases of hiatal hernia according to sex revealed that the condition occurred slightly more frequently among females than among males, in a proportion of 133 to 113. The condition is no respecter of age and may occur at any period of life, although it is much more frequent after the fourth decade. The average age of the above group was fifty-five years; the youngest patient was eight years of age and the oldest eighty-two years. All but nineteen of the patients had clinical symptoms which could be directly attributable to the hernia. It was impossible, from the clinical history alone, to distinguish between the three types of hiatal hernia.

As Morrison[6] and his co-workers pointed out, the first and by far the most frequent symptom associated with hiatal hernia is pain

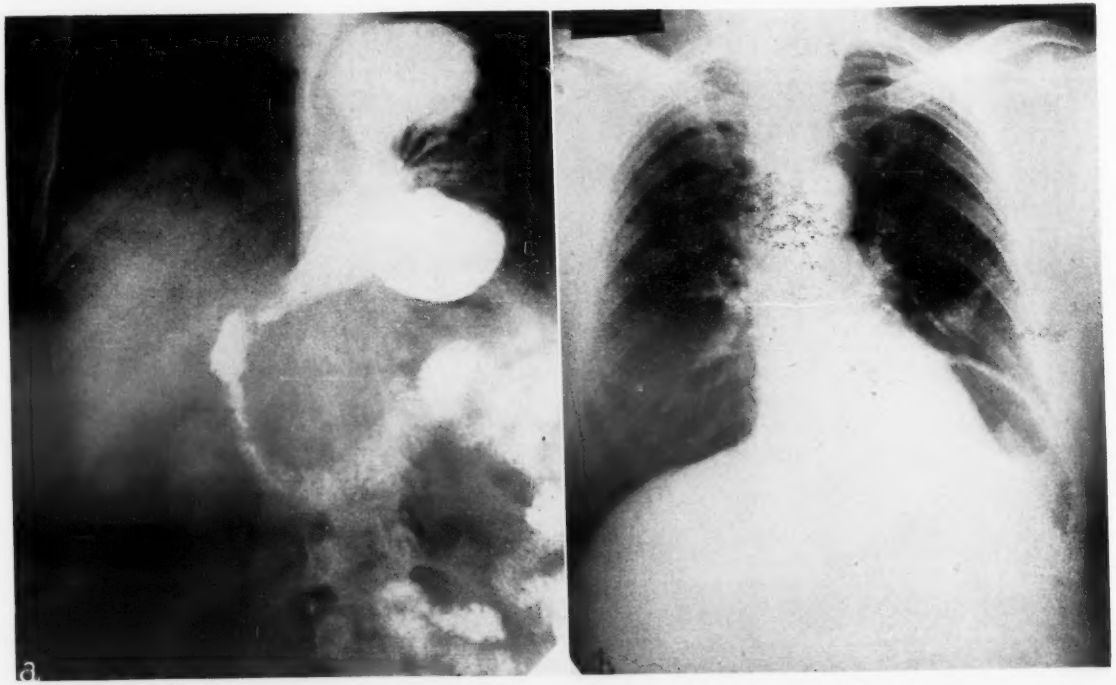

Fig. 2. *a*, Para-esophageal diaphragmatic hernia. *b*, Roentgenogram of the thorax in the same case.

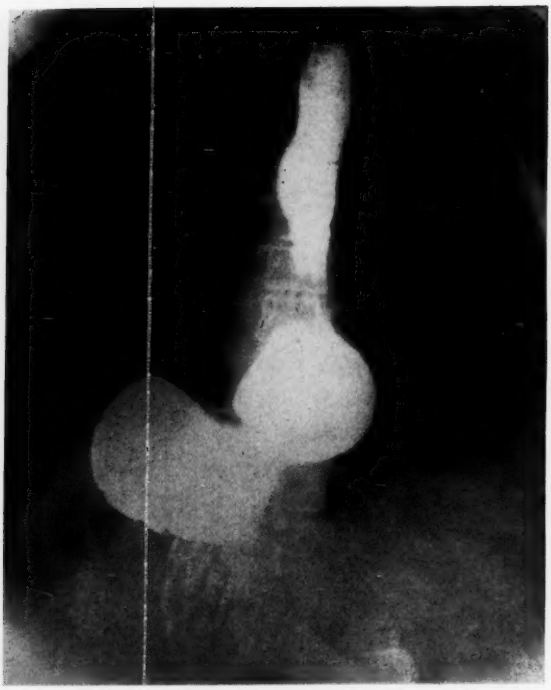

Fig. 3. Sliding type of hiatal hernia.

situated under the lower end of the sternum or high in the epigastrium. At the onset, this is usually of a mild nature and intermittent in character, usually coming on while, or shortly after, eating. The attacks generally tend to increase in severity, dependent on the degree of incarceration and the associated spasm of the esophagus and diaphragm. The pain frequently is projected through to the back or about the costal margins and, less frequently, to the left shoulder. Usually it is associated with gaseous eructation. Many patients obtain relief through the use of small doses of soda or milk, and, consequently, the symptoms may be misinterpreted for those of peptic ulcer. The pain may become more severe and be associated with vomiting and belching and may simulate closely cholecystic disease. Harrington[7] has pointed out that many of these patients undergo

Fig. 4. Cicatrical stricture of esophagus with small hiatal hernia.

unnecessary operation on the stomach and gallbladder because of mistaken diagnosis. As a result of accumulation of gas in the herniated stomach, spasm of the diaphragm may occur with phrenic pain which is referred to the left shoulder and down the left arm, closely simulating angina. There is further likeness to angina in the fact that the incarcerated stomach may be distended with gas and may produce increased intrathoracic pressure with resultant tachycardia, palpitation and dyspnea. The above symptoms frequently are aggravated when the patient assumes a recumbent position.

Dysphagia was present in approximately 20 per cent of our cases. This varied from a sense of slight intermittent obstruction up to almost complete esophageal closure. Very frequently, dysphagia was noted at the outset of a meal, but if the patient was able to belch

Fig. 5. *a*, Large hiatal hernia with associated pharyngo-esophageal diverticulum. *b*, Large hiatal hernia with associated pharyngo-esophageal diverticulum with evidence revealed in roentgenogram of the thorax.

or vomit he could then complete the meal without difficulty. This symptom was present somewhat more frequently and with greater severity among patients who had congenital short esophagus with intrathoracic stomach than among those who had the other two types of hernia.

Bock, Dulin and Brooke[8] have called attention to the importance of a thorough search for hiatal hernia in any case of unexplained anemia. Thirty-two of our 246 patients gave a history of bleeding from the gastro-intestinal tract. The only symptom of which several patients complained was that of weakness, and on examination, these patients were found to be anemic, with evidence of loss of blood from the gastro-intestinal tract. Roentgenologic studies revealed the presence of hiatal hernia, and treatment directed toward this condition was followed by relief of the anemia.

The cause of bleeding in cases of hiatal hernia is not always apparent. Although, in some cases, a definite ulcer may be found in the hernia, usually at the site where the stomach is in contact with the hernial ring, this is not always true. Erosion may be found at the esophagogastric junction which may account for the loss of blood. In other cases, no definite cause for bleeding can be found, either grossly or microscopically, the bleeding apparently occuring by diapedesis (Fig. 3).

Strangulation of an incarcerated hiatal hernia with symptoms of intestinal obstruction is a rarity. This is owing to the fact that the stomach is a very muscular organ which would be difficult to strangulate. Further, the hiatal ring is usually of such size that it is not likely to produce obstruction. When symptoms of intestinal obstruction occur, usually some other viscus has slipped up through the esophageal hiatus, either with or without the stomach. When this occurs, there are all the usual symptoms of intestinal obstruction, with the addition of severe gastric hemorrhage. I have seen this complication occur twice, once with hemorrhage and once without hemorrhage.

Although the diagnosis of hiatal diaphragmatic hernia is dependent primarily on roentgenologic studies, I wish to call attention to the value of esophagoscopy in the study of the condition. In spite of the excellent articles dealing with esophagoscopy in the study of hiatal hernia, especially that of the congenital short esophagus with intrathoracic stomach, by Findlay and Kelly[3] from abroad and by Manges and Clerf[9] of this country, the subject has not received the attention which it deserves. Harrington,[7] who has had a most unusual experience in the surgical management of esophageal hiatal

hernia, feels that esophagoscopy should be carried out in every case of hiatal hernia in which operation is to be undertaken.

The esophagoscopic findings in cases of congenital short esophagus with intrathoracic stomach are fairly typical and constant. These consist of a short esophagus with narrowing of the esophagogastric junction and the finding of a dilated portion below this point lined with gastric mucosa, with no evidence of the normal diaphragmatic hiatus. The point of narrowing may be of two types. The first, and by far the more frequent, is the fusiform narrowing which may measure anywhere from 1 to 7 cm. in length. The other is in the form of a membrane which partially obstructs the lumen. Although Findlay and Kelly[3] found no evidence of ulceration or erosion at the point of narrowing, our experience was similar to that of Monkhouse and Montgomery,[10] and that of Manges and Clerf,[9] that ulceration does occur at this point and that it may simulate that of carcinoma. The third, or sliding type of esophageal hiatal hernia, at times, may resemble closely the congenital short esophagus with intrathoracic stomach. The esophagus in the sliding type, however, usually is more redundant and the point of narrowing of the esophagogastric junction is less pronounced and shorter. Erosion and ulceration may occur in these cases as well as in cases of congenital short esophagus and may lead to difficulty in differential diagnosis. When doubt exists as to the presence of hernia, the procedure outlined by Findlay and Kelly[3] is to be advocated. This consists of measuring the distance of the gastric mucosa from the incisor teeth. In cases of doubt, it is advisable to snip a piece of tissue at this point to be certain that it is gastric mucosa. It is also of value to examine it roentgenologically, with the esophagoscope in place, to determine the level of the esophagogastric junction in relationship to the diaphragm (Fig. 1b).

Bleeding, as previously mentioned, is a frequent accompaniment of esophageal hiatal hernia. When this is present, esophagoscopy is advisable to determine whether or not ulceration and erosion are present and, if so, how extensively. It is also advisable to determine whether or not any other lesion may be present to account for the bleeding.

Carcinoma at the cardia at times may offer a great deal of difficulty in diagnosis roentgenographically, and this is especially true, as Manges and Clerf[9] have pointed out, when associated with esophageal hiatal hernia. Fig. 6 represents a patient of this type and illustrates the importance of esophagoscopy to avoid possible unforeseen therapeutic problems.

Fig. 6. Carcinoma of cardiac end of stomach associated with hiatal hernia.

The possibility of a foreign body becoming lodged at the point of obstruction and obscuring the true underlying condition always must be considered. The following case is an example of such a possibility:

REPORT OF A CASE

CASE 1.—A man, aged 63 years, gave a long history of indefinite gastric distress with an occasional sense of dysphagia. Suddenly, a year before his admission to the Mayo Clinic, the dysphagia had become more severe. Roentgenographic studies made at his home were reported as giving evidence of carcinmoa of the esophagus. Esophagoscopy had been performed and a growth in the esophagus had been reported. The patient had continued to have increasing difficulty and, at the time of his admission to the clinic, he had lost forty pounds and had almost complete esophageal obstruction. Roentgenologic examination gave evidence of an irregular filling defect in the lower part of the esophagus, suggestive of carcinoma (Fig. 7).

Fig. 7. *a*, Esophageal obstruction produced by persimmon skins; patient had hiatal hernia. *b*, After removal of foreign body.

On esophagoscopic examination a tumor was found obstructing the lower part of the esophagus. A portion of tissue removed from the tumor was shown to be persimmon skin which the home physician, who had accompanied the patient to the clinic, recognized immediately. The tumor was removed piecemeal and was found to consist of persimmon skin and other vegetable fibers. Following its removal, the patient was found to have narrowing of the esophagus at the point of obstruction, with gastric mucosa immediately below, and the condition was interpreted as a congenital short esophagus with a partial intrathoracic stomach. This diagnosis was confirmed by subsequent roentgenographic studies. An interesting feature of the case was that the last time the patient had eaten persimmon was a year previously, just preceding the time when dysphagia increased in severity.

Although cardiospasm, eventration of the diaphragm, diverticulum of the lower part of the esophagus and diffuse spasm of the lower part of the esophagus may be associated with roentgenoscopic pictures suggestive of esophageal hiatal hernia, this is the exception rather than the rule. When doubt exists, esophagoscopy should be carried out.

One of the most difficult problems at the present time is to determine where the borderline exists between normal hiatal relaxation, to which Findlay and Kelly,[3] Schatzki[11] and Wagner[12] have called attention, and true esophageal hiatal hernia. It has been pointed out by Kirklin[13] that, if a roentgenogram is obtained during deglutition among normal individuals at the precise moment the bolus of barium is entering the stomach, a picture that closely simulates hiatal hernia may be obtained. In cases of true hernia such a picture is not transitory, but can be produced over a longer period of time. In case of doubt, esophagoscopy with removal of gastric mucosa, biopsy and roentgenographic studies with the esophagoscope in place will be of distinct value.

The problem of treatment in cases of esophageal hiatal hernia is dependent on the type of hernia and the severity of symptoms. In cases in which there is an absence of symptoms or in which the symptoms are of mild character, treatment is not indicated. When the condition is owing to congenital short esophagus with intrathoracic stomach, dilatation of the narrowing at the esophagogastric junction usually will be sufficient. I wish to point out, however, that if definite stenosis is not present at the esophagogastric junction and, especially, if there is an absence of ulceration, the condition may not be a true short esophagus, although the roentgenologic and esphagoscopic findings would seem definite. This is based on the fact that, in a number of cases in which dilatation did not relieve the patient and the symptoms were of such severity as to produce marked distress, we felt justified in exploring the esophageal hiatus. In a number of cases of this type, Dr. Harrington was able to bring the

stomach back into its normal position below the diaphragm with subsequent relief of symptoms and with no roentgenologic evidence of the hernia postoperatively. In cases of other types of hernia, if the symptoms are severe, surgical repair is the procedure of choice. The postoperative results of repair of esophageal hiatal hernia are, as a rule, very gratifying. Sixty-four of the 246 patients in my series were subjected to operation.

If the patient is very obese or an extremely poor surgical risk, simple reduction in weight may afford a great deal of relief. Interruption of the left phrenic nerve was carried out in thirteen of the cases of the series, with improvement in symptoms in seven cases. In the remainder, the symptoms were aggravated. We have come to feel, however, that this procedure probably is not advisable, as it may lead to postoperative relaxation of the diaphragm with consequent danger of increased difficulty.

In conclusion, it is apparent that the problem of hiatal diaphragmatic hernia is a subject that is deserving of our most careful consideration. Furthermore, esophagoscopy should be considered in every case because of its additional value in the proper treatment of the condition.

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LVIII

III. THE DIAGNOSIS AND TREATMENT OF MÉNIÈRE'S DISEASE

S. H. Mygind
AND
Dida Dederding

Copenhagen

As every treatment depends upon the correct diagnosis, we shall mention some of the most important diseases which may give rise to mistakes.

In many cases of middle ear suppuration, giddiness may occur as a result of collateral labyrinth edema, without there being any question of propagation of the inflammation proper to the labyrinth. Such conditions need not afford an indication for surgical intervention, and they may frequently be treated as Ménière's disease, from which they do not in reality differ.

In case of so-called middle ear catarrh and tubal stenosis it will frequently be detected, by mere questioning and sufficiently thorough examination of the patients, that they are subject to the same giddiness and the same nystagmus as Ménière patients. There is only a difference in the location of the morbid process. In the proper case it may often be beneficially influenced by the special Ménière treatment. The same applies to the so-called neurolabyrinthitis, which presents the same impairment of sound conduction and the same frequent combination with otoscopic changes, tubal stenoses and general symptoms.

This applies particularly to the syphilitic so-called neurolabyrinthitis. Thus, in only one out of thirty-nine cases did we find a pure disturbance of perception.[1] Otherwise the symptomatology does not in any day differ, apart from the Wassermann reaction, from the genuine Ménière disease, and there is a pronounced tendency of water retention.

The distinction may prove difficult in case of otosclerosis. The protracted, gradual, usually symmetrical development, the missing or faint vestibular phenomena are suggestive of otosclerosis in contradistinction to the erratic progress characteristic of Ménière's dis-

ease. However, an apparently typical otosclerosis with a negative Rinne test and very severe bass deafness may turn out to be Ménière's disease or syphilis, as shown by a distinct improvement of hearing subsequent to injection of salyrgan.

Apparently typical Ménière conditions may accompany or initiate a series of intracranial affections,[2, 3] the most frequent of which are epidemic encephalitis, disseminate sclerosis, cerebral tumor, and tumor of the acoustic nerve. In some cases it is impossible to say whether the condition should be termed encephalitis or arachnoiditis with specially pronounced ear symptoms, or Ménière's disease with particularly marked intracranial symptoms, and changes of the cerebrospinal fluid.

These patients must therefore be submitted not only to an otological but also to a very careful medical and neurological examination comprising ophthalmoscopy and, if possible, an examination of the cerebrospinal fluid.

In case of an acute attack it will as a rule be sufficient to confine the patient to bed in a dark room. He should receive as little medicine as possible. In case of very severe attacks we think that we have witnessed a favorable effect from subcutaneous injection of 0.5 mg. of atropin. With regard to bulbocapnin recommended by Berggren and the various remedies for seasickness, we are without personal experience.

As regards the treatment proper,[4] we have likewise given up administering much medicine. Even mild diuretics frequently give rise to nausea, anorexia and sleeplessness, and a powerful remedy such as salyrgan frequently leaves the patient in a subsequent state of fatigue and a tendency to recurring retention. Moreover, even after administration of small doses of but 1 cc., we have in isolated cases observed temporary hematuria and albuminuria.

Instead of stimulating the discharge, we have therefore proceeded to reduce the intake of fluid and, at the same time, the consumption of salt. It will often be practical to let the patient, before this is done, continue his usual mode of living for three or four days, meanwhile recording all that he eats and drinks. The information thus obtained with regard to his daily consumption of food and drink frequently is stupefying. Afterwards he is submitted to the water test. It depends upon the retention during the water test, the mode of living, and the work of the patient as well as the climate, how great a limitation of fluid will be required in the individual case. In case of retention of 250 to 300 g. we allow the patient an average

intake of 600 cc. of fluid, including drinks, vegetables and fruit. On the other hand we do not take into account the water contained in meat and bread. On the whole we do not aim at an exact dosation. What matters is that the patient is on the verge of thirst. This can be seen by the tongue, which must be dry with moist borders. The patient should not be tormented. If he has been used to very liberal intake of liquid it may be necessary to reduce the intake by degrees. In case of more considerable retention we reduce the intake to 500 g. and eventually to 400 g. of liquid. If the discharge by water test is normal we allow an intake of 800 g. of liquid, seldom more, unless there is a tendency to concretions in the urinary tracts. At the same time no extra salt is served with the meals which are, however, prepared in the kitchen as usual. Potatoes are, for instance, boiled with salt, and ordinary salt butter is served, but no salt is served with meat, tomatoes or eggs. Particularly salt food, such as anchovies, sausage, cheese, broth and most potted meat is avoided. Liquid is preferably given in the form of fruit. Dry fruit may be given freely. Care is taken that the diet does not become deficient in vitamins. Since milk is avoided as liquid, cheese as salt, and the consumption of fruit is likewise limited, it may become necessary, in order to prevent distressing deficiency symptoms, to give an additional supply of calcium.

If the patient's weight is too high, the calorie content of the diet is limited, if it is too low, he receives an additional supply of butter and cream, besides arsenic. The weight is controlled at suitable intervals, daily intake and output of fluid are measured.

It does not suffice, however, to empty the abnormal water depots in the ear and elsewhere by means of such a treatment. It is at least equally important to ward off those factors which give rise to retention, above all the capillary dysfunction in the tissues.

This it is endeavored to attain by stimulating the circulation in the skin, the muscles and the lungs, since it seems as though an improvement of the capillary function and the consequently ensuing, increased localized hyperemia has a tendency to spread to other parts of the body. Thus Hasselbalch, after irradiation of the anterior surface of the thorax with carbon arc light, observed a permanent rise of cutaneous temperature, i. e., an increased perfusion with blood, not only in the area exposed to the irradiation, but also for instance on the back of the patient.

We have apparently derived our best results from irradiation with carbon arc light (Finsen light) whose spectrum is very much

like the solar spectrum. The baths are given every other day, beginning with 10 minutes and cautiously increasing with individually varying dosage up to 60 to 75 minutes, about 18 baths altogether. For at last the pigmentation of the skin becomes so strong that the baths must be prolonged very much if they are to produce any effect.

Many other forms of physiotherapy may, however, be applied: Carbonic acid baths, sulphur baths, pine-needle baths, or natural sun baths. During the first baths the patient may develop increased tinnitus and sometimes giddiness. Gytje baths and incandescent light baths must be applied with great precaution, for these patients, owing to the bad skin function, often have difficulty in perspiring, and may experience nausea because of the inhibition of their temperature regulation. Moreover, some of these patients cannot endure a cold shower-bath, and may suffer collapse. However, this defective vasomotor function may, in a certain degree, be improved by cautious administration of alternate, warm and cold shower-baths (Scotch shower-bath), or by the patient's standing with his feet in warm water. Besides, it is one of the most important rules that a Ménière patient must never feel cold, whether during treatment or otherwise.

In his own home the patient can do with the so-called salt brush-bath: 5 kg. of coarse salt in an ordinary tub bath of 37° C. After five minutes' stay in the bath the whole body is brushed thoroughly with a soft brush and subsequently rinsed with cold or temperate water. The temperature of the bath may gradually be raised to 39° C. and its duration to 15 minutes.

If the patients present marked subcutaneous or muscular infiltrations, they receive general massage which, during the first days, may be very painful. Gymnastics are beneficial also. The effect of massage, as well as of baths, particularly of light baths, is increased, if the patient can take one-half to one hour's rest in a recumbent position afterwards.

On the whole it is essential not to overdo the treatment but to apply a suitable mixture of stimulation and rest during cautious training. A rest of one to two hours in the middle of the day in the open air as that applied in case of tuberculosis, will frequently be beneficial. Plenty of outdoor exercise, preferably in a highland climate, is commendable as a completion of the cure.

As to medication, we have previously mentioned that, in order to prevent calcium deficiency, it may be necessary to supply the patients with additional calcium just as they receive arsenic if their weight is too low. In the fairly frequent cases of anemia the

patients receive iron; on the whole the hemoglobin must continually be controlled. During the climacteric ovarian preparations will often be necessary. Thyroidin should only be given in case of true hypothyroidism. We have now and again used secale preparations on account of their presumed influence on the paralyzed capillaries. These are at other times presumably the seat of spasms. Therefore we have also tried acetylcholin, which in isolated cases seemed to be effective, particularly in cases without water retention and with good hearing. In these cases calcium preparations may also be tried, eventually as a calcium diuretin. However, with regard to these latter remedies, we must reserve judgment. On the whole, we administer as little medicine, including soporifics, as possible. In case of constipation, frequently aggravated in the beginning of the treatment, the patients receive paraffin oil, eventually cascara or laxative salt, in the latter case with an extra allowance of water.

Several years ago Göthlin suggested that a C-Avitaminosis was a frequent cause of Ménière's disease. Elmenhoff later tested the capillary resistance of Ménière patients brought into our clinic during a year. He found it on the whole somewhat lower than in a series of control cases. Many very pronounced Ménière cases, however, exhibited perfectly normal reactions and no history of vitamin deficiency in the food. We therefore assumed an avitaminosis to play but a comparatively small part. During last spring we, nevertheless, have had some very striking cases, so that we should recommend keeping an eye on this phase of the problem and always testing the capillary resistance, and eventually also the ascorbic acid content of the serum.

Finally there is the local treatment with regular catheterization of the eustachian tube, not only for the prevention of an eventual stenosis, but also for the mobilization of the auditory ossicles and in order to influence the content of the labyrinth via the fenestrae. That this is possible is evidenced by the previously mentioned nystagmus, which is frequently witnessed in connection with the catherization. Diathermy, the application of which should theoretically be well founded, has not yielded any results in our hands.

It will often be necessary to hospitalize the patient during the first month in order to acquaint him thoroughly with his disease, to provide him with sufficient rest and quiet and to keep a strict eye on his regimen. Afterwards he may be allowed to continue at home, his treatment eventually being modified somewhat though under constant control, above all with regard to weight and hearing. After the lapse of three to six months the majority of patients may be allowed to proceed to an ordinary mode of living. They will, however,

always be subject to recurrences after infections, particularly influenza, after over-exertion, during the puerperium and menopause. Frequently a prophylactic treatment, for instance, Finsen light, will be useful in the winter, which is the time of danger.

With the help of the above treatment it will, as a rule, be possible to cure the patients of their giddiness, or at least to reduce it to a minimum, and in many cases to obtain an improvement of their hearing. At the same time the general health of the patient undergoes a change. We have previously published the results derived from the treatment of 157 patients. On dismissal from the hospital, 150 of them had recovered from giddiness and spontaneous nystagmus. Also, the acoustic result must be deemed satisfactory. In more than half of the patients (85) the hearing had distinctly improved; in 27 patients, for instance, from whispering 0.50 m. to 20 m. Besides there are 47 patients whose hearing was either normal or so slightly impaired that this was not detected until they were submitted to function tests. Also the majority of the patients with such minimal reduction of hearing recovered entirely or almost entirely. In the remaining 24 cases (14 per cent) the hearing had not changed, or it had even decreased (in two cases).

But how long do these results last? We have been able to follow up 83 of these patients for three years or more; 67 of them (81 per cent) were permanently free from giddiness. Two of the remaining 16 had been free from giddiness and therefore thought they were safe in discontinuing their cure. Four incurred an attack after the lapse of two years. Two patients continued to suffer attacks, which, however, were not so severe as before. In only five patients the condition was unchanged, but it is worth mentioning that they all had too high a body weight and that they did not attend to their routine.

The acoustic result was as follows: In 29, i. e., about half of the 61 patients whose hearing had improved considerably during the first treatment, this improvement was permanent. In 24 patients the hearing was worse than on their dismissal, though better than at the time of their preceding admission to hospital. In this group there were only eight, i. e., 13 per cent, whose hearing was worse than on admission. Twenty of the patients with normal or minimally impaired hearing reported for after examination. The hearing of only one patient had diminished. The remaining patients presented normal hearing. Finally the hearing was unchanged in nine of the group of 12 patients in whom the first treatment was unsuccessful, in two cases it had improved, and in one case it was reduced.

Thus, the acoustic result must on the whole be termed satisfactory also.

We may add that this is not only due to inflation of the tubes. Among those 27 patients who were able after treatment to hear whispering at a distance of 20 m. instead of previously 0.50 m., two had not received inflation. In six patients inflation proved unsuccessful until they were submitted to general treatment.

Isolated cases defy all treatment, but in the majority of cases the bad result must be ascribed partly to inefficient control of the patient and partly to insufficient individualization of the treatment. What matters is to find out the combination of causal agents, exogenous and endogenous, physical and psychical, which are at the bottom of each individual case. Hence, the treatment of Ménière's disease claims great devotion on the part of the physician. On the other hand he has the satisfaction of being frequently able not only to help these ill-fated and often misunderstood patients, but also to become acquainted with a series of biological processes, the importance of which reaches beyond the confines of otology, as we shall endeavor to show in our concluding article.

(The final article of this series will appear in the December issue.)

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LIX

SINUS THROMBOSIS

PART IV

VENOUS THROMBOSIS

Thrombosis of the Superior Cerebral Vein

Cullen Ward Irish, M.D.

Los Angeles

Thrombosis of the superior cerebral vein without involvement of the superior longitudinal sinus rarely occurs. The first recorded case is by Dowman[121] in 1926. In his patient the symptoms were similar to those which developed in another patient after ligation of the rolandic vein in exploration of an inflammatory occlusion of the foramen of Monro.

The man, aged 37, had been in good health until a twitching sensation appeared in the right upper eyelid for five seconds, followed by an attack of dizziness lasting six or seven seconds which was so severe he had to hold to support to escape falling. The following three days, twelve to fifteen such attacks appeared daily. There was no actual eyelid twitching, it was simply a twitching sensation. Five days after the first appearance of this sensation, a feeling of numbness in the left hand gradually extended over the entire left arm and into the left side of the neck and face. Seven days later this sensation had involved the left side of the body and the left leg somewhat. The patient noticed that in spite of the numbness, sensation was only decreased in the involved left side, not absent. As his arm had begun to feel numb, awkwardness of movement developed in the affected extremity. There were several attacks of headache, more severe in the right parietal area. The attacks of dizziness ceased after the development of the numbness. The left hand and forearm were weakened and sensation was obtunded but slightly on the left side. There was astereognosis in the left hand, loss of joint sense in the left fingers, and of position sense in the left arm and leg. Fine and coarse cloth and slight differences in weights could not be differentiated in the left hand. The left arm and leg reflexes were exaggerated and the left abdominal reflexes were sluggish. At oper-

ation the rolandic vein was found to be large, hard and bluish black with compensatory dilatation of the neighboring veins and an excessive amount of fluid in the sulci.

Eight to ten days postoperatively, improvement which continued was noted in the left arm and leg. Three weeks and thirty-four days postoperatively there were no residual neurological manifestations except slight subjective numbness in the right hand, functions being resumed with re-establishment of the venous circulation.

A second case was reported by Waggoner[122] in 1928. A white male of 76 was admitted to the hospital with right hemiplegia. Two weeks before he had awakened unable to stand. There had been no unconsciousness or convulsions but he had felt dull. The right leg was paralyzed and the right arm was weak. There had been no facial palsy, no speech disturbance and no loss of sphincter control. Improvement had occurred, the right arm strength to almost normal, and some strength had returned in the right leg, but he was unable to stand on it. Occasional shooting pains appeared in the right leg. When admitted, the blood pressure registered 92/70. There were inco-ordination and loss of power on the right side, a questionable right facial weakness, and slight tremor of the tongue and hands. The right biceps and patellar reflexes were increased and the abdominal and cremasteric reflexes on that side were absent. There was no Babinski reaction. Some astereognosis was present on the right with no loss of tactile or temperature appreciation. Gradual improvement was occurring until one and a half months after his admission he died from bronchopneumonia.

At necropsy a large thrombosis of the left superior rolandic vein was found. All the veins in the middle portion of the convex surface of the left cerebral hemisphere were so dilated as to present the appearance of actual varicosities. The hard thrombus in the vein was one centimeter in diameter and four centimeters long. It did not extend into the superior longitudinal sinus. On coronal sectioning a plexiform honeycomb mass of thin walled blood vessels, the largest two centimeters in diameter and showing degenerative changes in the form of calcium deposits but no minute hemorrhages as described in the thrombosis of the vein of Galen, was found at the level of the superior surface of the corpus callosum immediately beneath the thrombosed vein. Waggoner suggested there might be some association between the presence of this typical plexiform hemangioma and the development of the thrombosis, but there was no proof of such a possibility, and the blood vessel mass was separated from the surface by a small amount of cortex.

Davis'[123] patient, a male of 42, showed slight weakness of the lower portion of the right side of the face and co-operated poorly in the examination due to his inability to maintain his attention. Anemia and low blood pressure were present. The spinal fluid was normal except for an increased globulin content. He developed right sided convulsions and right hemiplegia and hypesthesia. Astereognosis in the right hand and bilateral Babinski reactions were found present. At operation and necropsy extensive thrombosis of the left rolandic vein and its branches was found. There were small hemorrhages in and immediately beneath this vein. The author considered venous stasis as causative of the thrombosis, an acute infection in the form of "grippe" with diarrhea were contributory factors. Passive congestion appeared in the liver and spleen.

Following a study of the cerebral cortical veins in five fetuses and in ten adult brains, O'Connell[124] summarized his observations as follows: (1) The radiation of the cortical veins from the region of the insula is very well marked in the fetus, and persists. The superior members of this group approach the margin of the hemisphere at right angles, but their terminal portions, mostly, change direction before entering the longitudinal sinus. This condition appears during the later months of fetal life and is found present in the adult brain. (2) With the growth of the cerebral hemispheres there is a tendency towards separation of these vessels into a superficial middle cerebral system and a system of veins around the margin of the hemispheres—the superior and inferior cortical veins. The superficial middle system is composed of at least three veins and the inferior anastomotic veins usually number two, persisting as evidence of the original arrangement. (3) The direction of the veins is determined by the growth of the cerebral hemisphere, the anterior veins being directed posteriorly and the posterior veins obliquely anterior as they extend beyond the superior longitudinal sinus. The posterior inclination of the inferior veins also results with growth of the cerebrum. (4) One of the superior cortical vein series appears in each of the precentral, central and postcentral sulci and similarly a tributary of the superficial middle cerebral vein is found in each sulci which separate the opercula of the insula. (5) The lacunae laterales are absent at birth. Past middle age there are two, one on each side of the superior sagittal sinus. They serve as lateral extensions of the lumen of the superior longitudinal sinus over the supero-medial margin of the hemisphere in the postfrontal, parietal, and sometimes also the anterior occipital regions where the majority of the arachnoid granulations are found. The floor of each lacuna is everywhere carpeted by a layer of these granulations. Le Gros Clark[126] states

the arachnoid granulations are first seen at eighteen months of age, and after this they become more numerous and increase in size throughout life.

VASCULAR ACCIDENTS OF THE VENOUS SYSTEM OF GALEN

Bagley[127] considers hemorrhage from the vascular system of Galen results in a definite clinical syndrome. In three fatal cases he reported the clinical course was identical. There was an early sharp rise in temperature amounting to 3 degrees F. a few minutes after the accident in one case; a 5.5 degrees F. elevation ten hours afterward in another case, marking the beginning of a hyperpyrexia which continued throughout the course, and fever gradually increased to from 106 to 107 degrees F. in all cases at the time of death. The pulse and respiration were increased in all instances. In all cases muscular power was more or less affected and the reflexes were increased. The motor disturbance was chiefly spastic. Pupillary reactions were disturbed and the cerebrospinal fluid was bloody in all. In one patient a small temporal opening revealed intracranial tension was less than normal and the brain was almost shrivelled and easily compressible by a spatula. In the second case there was a space of at least one centimeter between the dura and cortex, demonstrating a lack of normal intracranial pressure.

The pathology shown at necropsy in two cases was so definitely limited to structures anatomically related to the vein of Galen and its tributaries that it is believed such a syndrome can be accounted for in the lesions described. The disturbances in the motor system, spasticity, weakness and increased reflexes, is fully accounted for by the pathological findings and hyperpyrexia, unconsciousness and low intracranial pressure could perhaps be better attributed to lesions of this part of the brain than those of any other, according to Bagley.

Eardley Holland[128] attributes distortion and rupture of the vein of Galen at the point where the straight sinus is formed as one of the results of trauma in the newborn. He found the vein of Galen ruptured twice in eighty-one examinations, but believes hemorrhages from some of the tributaries is a much more frequent accident.

Bedford[129] states that after occlusion of the great vein of Galen, no matter at what point on its course, collateral circulation is rapidly established. The choroid plexuses remain unchanged in their gross appearance. No indisputable case of hydrocephalus following thrombosis of these venous channels has been recorded. But sixty cases of primary thrombosis of the internal cerebral veins have been reported. Twenty-one of these occurred in infancy, and Ehlers and

Courville[130] have recently added five more with an intimate discussion of the entire series. Eight of the group were less than six months old and nineteen were under two years of age. They add a third group, that of combined—due to accumulated effects of both focal infection and a general disease—to the usual classification into the primary (marantic or antochthonous) and secondary (infections or septic) types. Gastro-intestinal disturbances, pulmonary, and acute infections are the most common predisposing factors in primary thrombosis of the intracerebral veins. Anemia is not likely as important a factor as in adults, and it is unlikely syphilis plays a role in the etiology. Tuberculosis may be contributory, at it was present in two of Ehlers and Courville's five cases.

The patient is usually seriously ill a variable time. In children the cerebral symptoms appear from three to twenty-one days after the onset of the vomiting and diarrhea in gastro-intestinal affections, during which period disturbed nutrition and dehydration have been produced. The cerebral symptoms were ushered in by convulsions in twelve of the twenty-six children. A few were drowsy in the beginning and the remainder presented sudden coma, opisthotonos or headache. Eleven were lethargic to comatose during their illnesses.

Where the thrombosis was an incident in the closing days of life, no cerebral symptoms were distinguishable. The temperature in thirteen was 104 to 106 degrees F., but in two 99 degrees F. was the highest registration. The pulse was rapid and irregular. Five of the eight in whom lumbar puncture was done showed an increased intracranial pressure. The facial veins were engorged in two, the eyelids were edematous in two others, and in two the optic fundi demonstrated increased pressure. Abnormal movements in the form of rhythmic convulsive contractions appeared in some, and rigidity of the extremities and entire body and trismus have been described. Paresis or paralysis was not observed in children, although it is common in adults. Nystagmus occurred in three and oculomotor disturbances in eight. Signs of meningeal irritation appeared in less than half of the patients. In five of the eight punctured, the cerebrospinal fluid was zanthrochromic. The cell counts were from normal to 1,020 per cubic centimeter, with lymphocytes predominating in three and polymorphonuclears twice. The sugar content was normal in two and reduced in two, in one of whom tubercular meningitis was present. All cultures taken were sterile. Signs of cerebral involvement were present from one to seven days before death.

The pathology is the result of obstruction of venous channels by thrombi and the variations depend upon the age of the patient, the length of the survival period, the point of the original obstruction, the rapidity and completeness of the obstruction and the influence of other possible lesions, e. g., tuberculous meningitis. The thrombosis may develop primarily in the internal system of veins, but more likely by a secondary extension into the internal venous system from the straight sinus. The lesion is a red softening of the area drained by the occluded vessel, affecting predominately the septum pellucidum, the corpus striatum, thalamus, the ventral part of the corpus callosum, the median aspect of the occiput or the median superior surface of the cerebellum. The ventricular fluid is usually blood stained or zanthrochromic.

VENOUS THROMBOSIS

Thrombosis of veins solely was demonstrated in ten instances, 2.45 per cent of all thrombotic processes investigated, and 10 per cent of all thromboses in the cerebral venous system. Five of the patients presenting this pathological condition were female and five male, one a negro. The age incidences were 18 days, 13 and 15 months, and 9, 17, 39, 50, 56, 57 and 77 years, averaging 29.9 years. Their hospitalization was from four hours to six days, except in the 18-day-old premature born in the hospital, in whom the time of the occurrence of the venous thrombosis was not definitely determinable, with an average of about nine days for the group. In the other patients survival was from one to six days, averaging four days.

Otitis media was present as an etiological source of the venous thrombotic process in seven, multiple pansinusitis of many years' duration in another, and in two, with sudden onset of coma, the causative factor was not determined.

The venous and pathological involvement in this group was as follows:

V. T. 1. Some adjacent softening was present about a thrombosis of the right anterior vein.

V. T. 2. With thrombosis of the left internal (terminal) cerebral vein, hemorrhage was found in the left basal ganglia.

V. T. 3. Right internal jugular vein thrombosis and meningitis with no brain pathology.

V. T. 4. Bilateral primary venous thrombosis had resulted in diffuse bilateral subarachnoid cortical hemorrhage.

V. T. 5. Thrombosis of the veins draining into the left lateral sinus, particularly the vein of Trolard and a second afferent vein just posterior to it.

V. T. 6. Thrombosis of veins with softening fronto-parietal region and sanguino-purulent meningitis following forty years chronic otitis media.

V. T. 7. Thrombosis of the choroidal veins with its extension into the veins of Galen and the straight sinus had produced marked bilateral basal ganglia softening.

V. T. 8. Subarachnoid hemorrhage had resulted from thrombosis of the right anastomotic vein. Thrombosis of the anterior cerebellar veins had been the origin of the intrapeduncular hemorrhage. The right cerebral veins emptying into the superior longitudinal sinus were thrombosed and a subdural abscess involved the right dorsolateral surface of the frontal lobe.

V. T. 9. A subdural abscess in the right cerebrum co-existed with thrombosis of the right anterior superior cerebral vein.

V. T. 10. Thrombosis of the veins of Galen and straight sinus with basal ganglia softening.

Brief summaries of those patients' illnesses and the pathological findings follow.

V. T. 1. A female 56 lived but 14 hours after her admission to the hospital following a fall to the floor that morning. She had been unable to speak for a few minutes, then the left lower extremity cramped and pain appeared in the left arm. Examination on admission disclosed paralysis of the left arm and leg, absent reflexes in the upper extremities and increased patellar reflex action but no pathological reflexes. The tongue protruded to the left and right arm and leg were in continuous purposeless movement. Thrombosis of the right anterior cerebral veins with some adjacent softening was the only brain pathology shown.

V. T. 2. The 18-day-old female premature infant had been delivered by Caesarian section of a mother with pulmonary tuberculosis and a tubercular hip. She had a bacillus coli infection and a 4-plus Wassermann reaction. The blood Wassermann on the baby was reported 4-plus and bilateral otitis media developed its first few days of life. The left terminal (internal cerebral) veins were found to contain a fresh thrombi.

Between the cerebral hemispheres was a hemorrhagic appearing area containing flecks of brain substance at the site of the left corpus striatum and extending from the head of the caudate nucleus almost to its tail. The left ventricle was filled with this bloody, wax-like material.

V. T. 3. Sore throat, nausea and vomiting, and painful, swollen joints had been present for several days in a 9-year-old boy when he was brought to the hospital. The right ear had been draining for a week following the onset of measles a week previously. Examination disclosed tender, swollen joints, tender calf muscles, bilateral cervical adenitis and the clinical symptoms of scarlet fever. The right ear continued draining and four days after his admission a left myringotomy resulted in drainage from that ear. The blood leucocyte count was 12,000 per ccm., 90 per cent polymorphonuclears the day after he was admitted and 15,066 per ccm. with 95 per cent polymorphonuclears the next day. The diagnosis of pneumonia was corroborated by roentgenological findings and death occurred the fifth day of hospital residence.

The brain was edematous and the meninges hyperemic, but no other brain pathology was present. The right internal jugular vein contained a septic thrombus, beginning two inches above the angle of the jaw, with superimposition of a rather firm postmortem clot in the vein. Advanced purulent right otitis media had involved the petrous portion of the temporal bone medially to a marked extent and the mastoid to a less extent. On the left the infection was confined to the middle. In the fascia under the superficial neck muscles of the right anterior portion, from the sternum to the angle of the jaw, was contained a pyemic abscess of undetermined origin, there being no evidence of a retropharyngeal abscess or a suppurating lymph gland.

There were a few subserous hemorrhages on the surface of the liver, large portions of which were pultaceous. The spleen was twice its normal size. Smears from the right ear disclosed the presence of streptococci and staphylococci, from the left ear pneumococci and from the jugular vein thrombus and neck abscess, chiefly streptococci and staphylococci.

V. T. 4. Following a fall to the floor with sudden unconsciousness, a male aged 77 was admitted to the hospital. With subsidence of the coma he found himself unable to speak, but he attempted to write. The blood pressure was 220/150, but no evidence of cardiovascular-renal or localizing cerebral vascular pathology was elicited.

He died the sixth day after the attack of unconsciousness. Brain examination revealed subarachnoid hemorrhage over the vertex and right temporal lobe. The right cerebral convolutions were very flat. Some subarachnoid hemorrhage was present over the right post central gyrus and left mid-posterior parietal lobe with some softening in the right mid-post parietal area. Primary venous thromboses had occurred in those involved areas.

V. T. 5. A 17-year-old boy had suffered from osteomyelitis of the right shoulder, and, seven weeks before admission to the hospital, left earache for a few days. When admitted he was vomiting and complained of loss of vision. Examination disclosed the presence of right homonymous hemianopia, slight elevation of the nasal sides of the optic disks, right facial weakness and increased reflexes on the right side. Pneumococci were found in smears made from the spinal fluid. The left temporal lobe was unsuccessfully explored for abscess the day of his admission and again four days later, with death occurring the next day.

At necropsy there was found thrombosis of the veins draining into the left lateral sinus, particularly the vein of Trolard, and a second afferent vein just posterior to it. Pneumococcic meningitis and left mastoiditis and otitis media were present.

V. T. 6. A white male of 57 gave a history of chronic left ear discharge for forty years. An attack of coryza had begun six days before he entered the hospital with acute left earache and severe left fronto-temporal headache persisting for four days. Chills and fever to 103 degrees F. had been present for two days and projectile vomiting the twenty-four hours before he was admitted. The left mastoid was slightly tender, the neck stiff and the left optic disk slightly elevated. Kernig and Brudzinski reactions were present and the reflexes on the right side were greatly exaggerated. The white blood cell count was 14,400 per ccm. with 81 per cent polymorphonuclear leucocytes, 10 per cent mononuclears and 9 per cent lymphocytes. The clear spinal fluid was under 200 mm. of water pressure, with slightly increased globulin and normal sugar content. It contained 67 cells per ccm., 40 per cent lymphocytes and 27 per cent polymorphonuclear leucocytes. Two days later the spinal fluid pressure was over 400, globulin content was 3-plus, there was no reduction of Benedict's solution with 30 drops of spinal fluid and 21 cells; 14 per cent polymorphonuclear leucocytes and 7 per cent lymphocytes per ccm. were present. It was questionable about the rise of spinal fluid in the manometer with pressure on the left jugular vein. Gram

Fig. 8. V. T. 4. Subarachnoid hemorrhage from venous thrombosis.

negative bacilli, probably bacillus influenza, were found in the spinal fluid smears. When a mastoidectomy was performed the mastoid area was found to be sclerotic with much granulation tissue present in the middle ear. Following the operation the temperature elevated and the pulse slowed. The patient was aphasic and presented an almost complete spastic right hemiparesis. The left pupil was larger than the right. Death occurred the next day, the third day after his admission.

A gray yellow purulent subarachnoid exudate was present, most marked over the inner surfaces of the occipital lobes and over the left parieto-temporal-occipital region, where it was sanguinopurulent. The base of the brain, particularly over the pons and optic chiasma, was covered with recent hemorrhage which extended over the left temporo-parietal-frontal area, where the veins draining this region were thrombosed with cortical softening underneath. The inferior anastomotic and sylvian veins were thrombosed with the recent hemorrhage in the sylvian fissure, having resulted from rupture of the sylvian vein. The left lateral sinus was unusually small, at no

point being more than 4 mm. in diameter, but neither it nor any of the venous sinuses were thrombosed. The radical left mastoidectomy had created a single cavity. The right middle ear and mastoid and nasal sinuses were negative. Smears from the brain showed the presence of numerous gram positive rods-bacilli influenzae.

V. T. 7. Another baby, a male 13 months of age, was admitted to the hospital in coma and lived but four hours. Pertussis had been present for three months and bronchopneumonia for two weeks. Otorrhea had developed four days before he was brought to the hospital. The spinal fluid pressure was low and 1,020 lymphocytes per ccm. were present. Smears from the ears showed bacillus influenza and diplococci. Coronal cerebral sections disclosed marked softening of the basal ganglia bilaterally and thrombosis of the choroidal veins extending into the veins of Galen and the straight sinus.

V. T. 8. A female of 39 had suffered an influenzal infection four weeks before she was brought to the hospital in semi-stupor. She had remained in bed six days, then had worked until eight days ago, when severe earache in the right and then the left ear appeared. Two days before her hospital entrance severe headache, backache and deafness developed and the next day she was stuporous. At examination she was found to be almost completely deaf and marked rigidity was present. The pulse rate was 110, temperature 104.4 degrees F. and respirations were 24 per minute. The right membrani tympani was red and bulging but no mastoid tenderness was present. The right patellar reflex was increased and bilateral Brudzinski reactions were elicited. The spinal fluid pressure was over 400 mm. of water, globulin was increased, 40 drops of fluid failed to reduce Benedict's solution, and 13,000 polymorphonuclear leucocytes per ccm, were present. Smears from the fluid showed pneumococci. She survived but 34 hours after entering the hospital.

The brain examination showed foci of subarachnoid hemorrhage about the frontal parietal and temporal lobes. A subdural abscess arising from the right tegmen tympani extended forward, the exudate involving the right sylvian fissure and the dorsolateral surface of the right temporal lobe. The hemorrhagic foci had originated from the thrombosed right anastomotic vein. Early septic meningitis was shown in the arachnoid at the base and over the anterior margins of the cerebellum. Small hemorrhagic foci lie near the left middle cerebellar peduncle and some, but less, in the same location, on the right side. The regional anterior cerebellar veins were thrombosed.

V. T. 9. A negro aged 50 gave a history of "sinus trouble" for years with a recent exacerbation seven weeks before he was admitted to the hospital. There had been intermittent pain and swelling in the frontal sinus for several weeks and two operations upon them, the last three weeks ago. Headache, nausea and vomiting had been present for two days and the morning of his admission right-sided convulsive movements with coma since. The clinical temperature was 103.4 degrees F., pulse 152 and respirations 44. An irregular, swollen and slightly fluctuant area was present over the left frontal region. All extremities were moved voluntarily, but the right less ably than those on the left. He was unable to speak. The pupils were small and irregular but equal. The fundal veins were full and tortuous. Babinski and Gordon reactions were elicited on the right side and bilateral Kernig reactions. Neck rigidity was moderate. Death occurred the third day of his hospital residence.

At necropsy a large, irregular, loose sequestrum from an osteomyelitic process was present in the left frontal region, from almost the midline to the temporal and 5 cm. from the supra-orbital ridge. The right frontal bone was roughened and its periosteum adherent. The accessary nasal sinuses were normal. Subdural abscess formation was extensive over the right cerebrum and slight on the left side with some localized extradural involvement in the left frontal region. The right anterior superior cerebral veins were thrombosed. No cerebral pathology was shown in the coronal sections.

V. T. 10. Diarrhea and vomiting had been present in a 15-month-old female babe for three weeks and convulsions for four days when she was brought to the hospital. The temperature registered 103.2 F., pulse 140 and respirations 40. Tetanic twitches and generalized spasmodic movements occurred frequently in the presence of coma and passive movements were resisted, but after forced motion the position was retained, the head bent between the knees being maintained in that position. Bilateral Babinski reactions and ankle clonus were elicited. Bilateral myringotomy produced no discharge. The spinal fluid pressure was 350 mm. of water with normal Queckensted response twice. The cell counts were 118, with 74 polymorphonuclear leucocytes at one puncture and 159 with 59 polymorphonuclears at the second examination. The sugar content was normal, globulin content 2 and 3 plus and a 4 plus Levinson reaction was reported. The white blood cell count was 30,300 per ccm. with 43 per cent polymorphonuclear leucocytes, 49 per cent lymphocytes and 8 per cent mononuclears. The blood calcium and phosphorus contents were normal. Bilateral myringotomy repeated

Fig. 9. Venous thrombosis with resultant extensive softening and moderate hemorrhage in both cerebellar hemispheres (R. B.).

resulted in pus drainage from both ears. The temperature was elevated to 108 degrees F., where it was maintained for two and one-half days, with the pulse increased to 170 and the respirations to 60 to 74. Death occurred the third day after admission.

A firm, red thrombus was found in the vein of Galen and straight sinus and the other posterior fossa sinuses contained some red thrombotic material. There was extensive softening of the basal ganglia bilaterally. Both middle ears contained yellow, purulent material, twice as much in the left as in the right. Most of the principal branches of each pulmonary artery were occluded by firm, pale thrombotic material.

After completion of the survey of 1,000 instances of vascular encephalopathy which included the 98 of sinus and venous thrombosis here presented, two further cases have been added with photographs of the pathology displayed.

VENOUS THROMBOSIS WITH MASSIVE BILATERAL CEREBELLAR AND BILATERAL BASAL GANGLION DIFFUSE HEMORRHAGES

R. B., a 38-year-old Mexican, was brought to the hospital in coma, cyanotic and breathing stertorously. He had been well until the night before, when he suddenly felt ill, had a chill and vomited

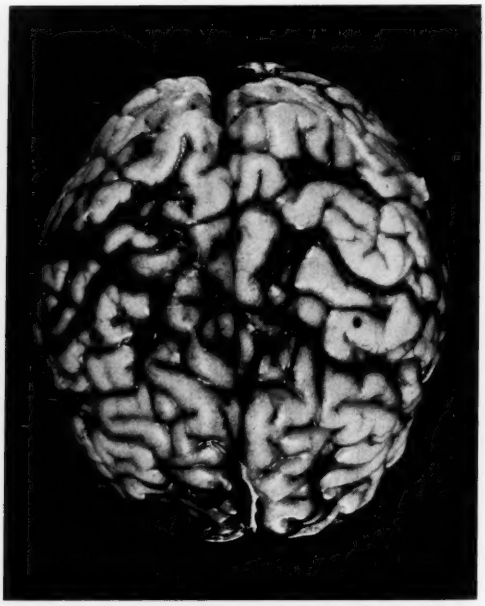

Fig. 10. Thrombosis of the superior cerebral veins with secondary subarachnoid hemorrhage, in a one year old baby (J. S.).

once. During the night he was restless and perspired freely. The following morning he talked irrationally and acted as if he were at his work. About 8:00 a. m. he became quite suddenly unconscious and entered the hospital six hours later, where he expired within five hours. During his observation in the hospital, intermittent tonic convulsions occurred. The pupils were small and did not react to light. Bilateral Babinski reactions were elicited but the deep reflexes were normal. The cerebrospinal fluid, under a very low pressure, was thin, bichromic and contained 50 red blood cells per cc. The urine contained 3 per cent sugar, the blood 333 mgm. of sugar per 100 cc. and gave a CO_2 combining power of 48.

The brain weighed 1,300 grams. The cerebral vessels were thin walled and normal in appearance. Coronal sections showed numerous hemorrhages, about equally distributed throughout both cerebral hemispheres. There was an area of hemorrhagic softening in the left mid-cerebrum. In the posterior third of the lenticular nuclei

Fig. 11. V. T. 10. Thrombosis of the central vein of Galen with resultant softening in the basal ganglia bilaterally.

petechial hemorrhages were uniformly distributed on each side in one section. The lateral ventricles were moderately dilated.

The pons and medulla were normal in appearance. Sections through the cerebellum showed extensive softening with moderate hemorrhage in both hemispheres, especially marked in the posterior portions. In the left lobe about the posterior half was involved and on the right side slightly less. At the tip of the left cerebellar tonsil and posterior to it there was small quantity of coagulated blood. The tonsil was slightly softened.

Microscopically several large vascular areas were seen in the meninges engorged with blood. Several small areas appeared in the brain substance some distances from the main hemorrhages. The microscopic diagnosis was venous thrombosis with hemorrhage, but no thrombosed veins were seen grossly.

Healed pulmonary tuberculosis and anthrocosis was present. Tuberculosis of the tracheobronchial lymph nodes was healed and chronic adhesive pleurisy was shown.

Chronic alcoholism and probable clinical diabetes mellitus were also contributory conditions.

CHART OF TEN CASES OF VENOUS THROMBOSIS

Serial No.	Autopsy No.	P. F. No.	Initials	Color	Sex	Age	Thrombotic Involvement Page
1	1506	194-927	A. L.	White	F	56	Right anterior vein
2	6475	100-108	B. N.	White	F	18 da.	Left internal cerebral
3	6507	103-465	T. B. Jr.	White	M	9	Right internal jugular
4	8341	197-012	Z. G.	White	F	77	Bilat. mid. cerebral veins
5	8742	189-279	H. C.	White	M	17	Vein of trolard
6	9134	89-796	I. B.	White	M	57	Inferior anastomotic vein with hemorrhage
7	9655	193-066	E. D.	White	M	13 mo.	Bilat. choroidal
8	9842	272-690	R. B.	White	F	39	Right anastomotic and bilat. ant. cerebellar veins
9	10225	290-369	O. J.	Black	M	50	Rt. ant. cerebrals
10	12113	328-795	S. P.	White	F	15 mo.	Vein of Galen Straight sinus

THROMBOSIS OF THE SUPERIOR CEREBRAL VEINS, PROBABLY SECONDARY TO THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS, WITH SECONDARY SUBARACHNOID HEMORRHAGE

J. S., a one-year-old baby, was admitted with rapid but not labored breathing following measles. The temperature registered 102 degrees F. during the first day, but the next two days varied between 102 and 104 degrees F. Breathing became more labored. The chest was filled with rales and presented general dullness. Throat culture was negative for diphtheria but the autopsy smear of the trachea was positive. Death occurred the sixth hospital day from bronchopneumonia.

The fixed brain was studied, but unfortunately the dura had been discarded, though there was, undoubtedly, a thrombosis of the superior longitudinal sinus and possibly of the other sinuses also. Otitis media had been present bilaterally and rather marked and extensive retrograde thrombosis into the venous channels likely had been present.

Coursing over the dorsolateral surfaces of both cerebral hemispheres were found a number of large engorged and partly thrombosed cerebral veins extending up to and terminating abruptly at the superior longitudinal sinus. It seemed likely that the thrombosis

started in the superior longitudinal sinus. In the right frontal region above and behind the right frontal pole appeared a local subarachnoid hemorrhage measuring 3.5 x 2.7 cm. in its greatest transverse diameters. This was due to rupture of some of the thrombosed veins in this region which showed considerable clotting in their lumen. Another blood clot filled in the sulci over the right parietal region.

1930 Wilshire Boulevard.

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LX

SPONTANEOUS CEREBROSPINAL RHINORRHEA

STANTON A. FRIEDBERG, M.D.

CHICAGO

AND

THOMAS C. GALLOWAY, M.D.

EVANSTON, ILL.

In a recent review of the subject of cerebrospinal rhinorrhea, Cairns[1] divides his cases into four groups: those occurring in the acute stage of a head injury, those occurring as delayed complications of a head injury, those produced during an operation on the cranium or the accessory sinuses and cases of spontaneous cerebrospinal rhinorrhea. (We were interested to note that in every case of cerebrospinal rhinorrhea associated with intranasal ethmoid surgery, the middle turbinate bone had been removed in the course of the operation. We found no instance of cerebrospinal rhinorrhea occurring as a result of a procedure in which the middle turbinate was left in situ and the ethmoid exenteration carried out lateral to this landmark.)

Among the cases of spontaneous cerebrospinal rhinorrhea belong those instances occurring with hydrocephalus associated with slowly growing intracranial tumor in any situation. An additional inclusion in this latter group should be those cases developing without any history of accident or of any predisposing cause. These are by far the least commonly encountered. The escape of cerebrospinal fluid in such spontaneous cases may occur along the sheaths of the olfactory nerves and Wagner Juaregg has recently suggested that sneezing may precipitate such a condition.[2]

It is well recognized that a patient with cerebrospinal rhinorrhea leads an hazardous existence and that the threat of meningitis is ever present. The various therapeutic measures suggested and employed have been numerous but have been concerned chiefly with cases having a traumatic basis.[3, 4, 5] There has been a general tendency to avoid all intranasal manipulation and to warn others concerning the dangers of such procedures. Fox, however, treated a patient with spontaneous cerebrospinal rhinorrhea by the local intranasal application of silver nitrate and obtained a permanent cessation of the discharge four months after instituting therapy.[6] We are reporting another successful case treated, in a modified way, along similar lines.

REPORT OF A CASE

CASE 1.—M. D., a white female, aged 38 years, was first seen in the outpatient clinic of the Department of Otolaryngology at Cook County Hospital in January, 1937. She had been disturbed for four months by a continuous watery nasal discharge which became more noticeable upon tilting the head forward. The secretion was clear, colorless and odorless, had a salty taste and did not stiffen the handkerchief upon drying. It was necessary for the patient to sleep on her side because the discharge ran into the throat when she lay on her back. The symptom dated directly from the termination of a severe head cold four months previously. There were no nasal or general manisfestations of allergic nature. The patient had had no headache nor dizziness, her vision had not changed and systemic inquiry was entirely negative.

Examination: The patient's nutritional status and development were excellent, the temperature, pulse and respiratory rates normal. The blood pressure was 120 systolic and 90 diastolic. The pupils were round, equal and reacted to light and in accommodation. The fundi were normal. A clear watery fluid could be seen escaping from the right nasal fossa. The rate of flow was approximately two ounces per hour. The nasopharynx was well visualized and presented no abnormalities. The remainder of the local examination, the general physical findings and a complete neurological study were entirely negative.

Chemical examination of the nasal secretion, collected by holding the head well forward, disclosed 6.5 mg. urea nitrogen, 60 mg. sugar and 795 mg. chlorides per 100 cc. Roentgenograms showed no skull abnormality and no apparent defect in the cribriform plate. The blood Wassermann test was negative. The blood count was within normal limits. No increase in spinal fluid pressure was noted on lumbar puncture, chemical examination corresponded to the above and, in addition, a negative Wassermann was obtained. There was one cell present. A diagnosis of spontaneous cerebrospinal rhinorrhea was obvious.

In an attempt to locate the site of fluid escape through the cribriform area, 5 cc. of methylene blue was introduced into the spinal canal and the foot of the bed elevated. No coloring of the nasal secretion was noted for 24 hours following this procedure.

Neurosurgical opinion did not favor an intracranial repair of the suspected defect. In March, 1937, we decided to attempt a simple intranasal measure in order to arrest the flow of spinal fluid. Accordingly, fluorescin was applied over the surface mucosa of the right olfactory sulcus, after which the fluid could be seen dropping from a region corresponding roughly to the junction of the middle and anterior one-third of the middle turbinate bone. A small cotton applicator saturated with 20% silver nitrate solution was held in this one area momentarily. One week later the discharge had markedly diminished and in 12 days it had entirely ceased. There was no subsequent recurrence and the patient has been completely symptom-free for 14 months. No headaches have been noted and no disturbance of smell resulted from the local application.

COMMENT

It is imperative that a sharp differentiation be made between the traumatic and spontaneous types of cerebrospinal rhinorrhea and that all available diagnostic measures be employed in an effort to establish the etiology of the so-called spontaneous cases. These measures should include serological tests,[7] roentgenograms of the skull and study of the eyegrounds,[8] in addition to complete physical, neuro-

logical and laboratory examinations. The most important point, however, is the necessity for not overlooking a case and in this connection it should be re-emphasized that examination of the fresh nasal discharge is essential because of the tendency for the chemical constituents to undergo alterations upon standing.

Having established the diagnosis of true spontaneous cerebrospinal rhinorrhea and being unable to locate any pathological process to account for the condition, it would seem fair to assume that the fluid is escaping along the olfactory nerve sheaths. The judicious application of silver nitrate solution to the appropriate area in the above case was sufficient to effect a permanent cessation of the discharge in 12 days.

Sir St. Clair Thomson has stated that some of these spontaneous cases will subside, temporarily or permanently, even without interference.[9] Others respond to bed rest with a series of lumbar punctures, although it would seem that such cases must have, as their basis, some form of intracranial hypertension. This recommendation, however, is certainly worthy of trial.

We do not feel that it is necessary, or even advisable, to swab the entire roof of the nose, as practiced by Fox. The use of fluorescin in the olfactory sulcus is of material aid in locating the approximate source of the fluid leak. The application of silver nitrate solution, with attention to asepsis, does not appear to us to entail any appreciable hazard in dealing with the small type of perineural dilatation which, in all probability, accounts for the escape of fluid in this particular variety of spontaneous cerebrospinal rhinorrhea.

122 South Michigan Ave.

636 Church St., Evanston.

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The Scientific Papers of the American Bronchoscopic Society

LXI

BRONCHIOGENIC CARCINOMA: A CASE REPORT*

HENRY B. ORTON, M.D.

NEWARK, NEW JERSEY

We do not have, or seldom obtain permission for autopsies in bronchiogenic carcinoma of the lung. There are many obscure factors concerning these growths. Usually nowadays the final diagnosis of cancer of the lung is made bronchoscopically, and, as Clerf has pointed out in a collection of data, unfortunately, diagnosis is late. This being so, little interest is shown in following up by autopsies the physical findings. Therefore, neither the extent of the lesion nor metastasis is recorded. I feel much valuable data is lost.

Metastases are curious, as for instance secondary tumors of the heart by contiguity, or via lymphatics, vena cava, pulmonary veins and by endocardial implants, and via coronary arteries. These are rare conditions.

Direct nonmetastatic invasion of the heart from contiguous lung growth is rare and the extent to which the heart may be supplanted by carcinoma without apparent serious disturbances is astonishing, as the pictures of this case show. Secondary tumors of the heart from the left side is a source of metastasis elsewhere, as we have in this case, to the liver.

This patient apparently showed little evidence of cardiac trouble, and the tremendous extent of the involvement warrants, I believe, the reporting of this case

*Presented before the Meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

REPORT OF A CASE

CASE 1.—R. J., aged 62 years, male, white; nativity, Ireland; occupation, moulder, was admitted to the Essex Mountain Sanitorium July 22, 1937, for observation.

Family History: His father died at 60 years of age as the result of an accident; his mother died at the age of 50 years from cholera morbus. There was no history of tuberculosis or cancer in the family.

Personal History: Patient had always enjoyed good health until December, 1936, when he had a continuous cough with expectoration. One month later he began expectorating a little blood accompanied with dyspnea, night sweats and progressive loss of weight (40 pounds in six months).

Physical Findings: Fingers clubbed, teeth bad, nose and throat negative; blood pressure 110/78; sputum negative for tubercle bacilli; blood Wassermann negative; urine negative.

Auscultation—left anterior—cavernous breathing with expiratory wheeze throughout on the right side; left base posterior—decreased breath sounds.

Blood picture: Erythrocytes 3,160,000, leucocytes 19,000, polymorphonuclears 87 per cent, hemoglobin 49 per cent. Chest aspirated, obtaining a thick fluid, which was negative for tubercle bacilli.

Localizing symptoms: Had pain in left chest, slight hoarseness, dyspnea, cough and expectoration, night sweats and loss of weight. Eleven examinations of sputum were negative for tubercle bacilli.

Roentgenograms were taken with the report as follows: "Right side emphysematous. Left side hazy throughout with no aeration of lung visualized. Atelectasis entire left lung. Trachea pushed over to left, interspaces narrower on left side, heart not visualized. Diaphragm elevated on left side to the ninth rib."

Bronchoscopy: The patient was referred to me for diagnostic bronchoscopy August 6, 1937. The conditions found on bronchoscopy were as follows: "On coughing, the carina compresses itself, rough area present at region of upper lobe bronchus on left side. Below the left upper lobe bronchus the main bronchus was narrowed by a growth partially closing same. A specimen was removed for biopsy and sent to the laboratory for histological study. The bronchoscopic picture was characteristic of malignancy."

The report of biopsy was bronchiogenic carcinoma, grade III.

The patient continued to go down hill and expired November 14, 1937, about six months after admission.

An autopsy was performed and the following is the report:

Thorax: Many adhesions on left side, none on the right.

Lungs: Right lung was soft, emphysematous, occupying entire chest and tended to lap over into the left chest. No pathological findings on sectioning this. Left lung adherent throughout, pus in isolated areas.

Heart: Pushed over to left more than usual. The heart and lungs removed together. The right main bronchus normal. The left main bronchus was completely occluded by a bronchiogenic carcinoma. This growth had invaded the parenchyma of lung so that the upper third of lung was involved. In the center of this involvement the lung had undergone a gangrenous process, so that it was completely broken down and there were several small cavities which were filled with necrotic lung tissue. The remainder of lung was shrunken and deformed. There apparently had occurred a great deal of fibrosis in addition to many bronchiectatic cavities.

Pericardial Sac: This had become completely obliterated, dense fibrous pericarditis. Heart normal in size, its surface nodular, so that from external appearance it was impossible to tell whether the myocardium had been affected or whether growth was merely on the visceral pericardium.

When the left lung was sectioned, it revealed that the entire myocardium and pericardium had been involved in the metastatic process. In addition, the myocardium was completely replaced by this spread from the lungs. It was apparent that in these areas the spread must have been by contiguity replacement of normal muscle by new growth.

The aorta compressed in transverse position.

Liver: Two metastatic growths the size of marbles.

The gall bladder, pancreas, spleen, kidney, stomach, intestines and bladder negative.

Lymph Nodes: No enlargement of axillary, mesenteric or supraclavicular.

DIAGNOSIS

1. Bronchiogenic carcinoma with invasion of parenchyma.
2. Metastatic involvement of heart.
3. Metastasis to liver.

(Drs. Taffet and Lomhoff.)

I am deeply grateful to Dr. Harman and Dr. Costellano of the Essex Mountain Sanitorium for their kindness in permitting me to show these specimens.

24 COMMERCE STREET.

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LXII

A CASE OF DYSPHAGIA DUE TO AN OSTEOCHONDROMA OF THE CERVICAL SPINE—OSTEOTOMY—RECOVERY*

SAMUEL IGLAUER, M.D.

CINCINNATI

Exostoses of the spinal column are frequently encountered, but seldom are so situated or attain a size sufficient to cause a narrowing of the esophagus by external pressure. The absence of symptoms in many of these cases may be explained by the mobility of the esophagus which is easily displaced by mediastinal tumors or deformities of the spine. In this connection the esophagus may be likened to a bowstring with two definite, fixed points of attachment, namely, at the cricoid cartilage and at the diaphragm; and clinical records have shown that when an exostosis does cause dysphagia, it is situated near the more immobile parts of the esophagus.

In the literature I have found records of six cases of vertebral protuberances causing difficulty in swallowing. Zahn[1] reported two cases, the first in 1905. His patient, a woman 36 years old, suffered from malnutrition and intermittent dysphagia. The difficulty in swallowing had been aggravated during pregnancy. The case finally came to an autopsy. An ecchondroma was found between the bodies of the eighth and ninth thoracic vertebræ. The esophagus was somewhat indented at the site of the tumor, and diffusely dilated above this point. Zahn was of the opinion that the esophagus had been subject to compression between the chondroma, the aorta, and the diaphragm when the latter was in an elevated position. In the following year the same author reported a second case with somewhat similar findings.[2]

Pincsohn's[3] patient died shortly after an esophagoscopy. At autopsy a perforation was found in the esophagus with bony tumors on the bodies of the ninth, tenth and eleventh thoracic vertebræ, with corresponding dents in the esophageal wall. Pincsohn attributed the dysphagia from which the patient had suffered to compression of

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*From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

Read at the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

the lower end of the esophagus between the spine, the aorta, and the diaphragm, as in Zahn's cases.

Mosher[4] reported two cases of difficult swallowing due to exostoses of the cervical vertebræ. In the first case, the x-ray showed two short spurs on the bodies of the fifth and sixth vertebræ. The second patient complained of solid food sticking in her throat, and had an exostosis of the bodies of the sixth and seventh cervical vertebræ. In the discussion of these cases Dr. Greene mentioned the case of a student who had exostoses on the bodies of the sixth and seventh vertebræ.

REPORT OF CASE

CASE 1.—*History:* Sept. 30, 1937, R. B., a white male, aged 68 years, complained of difficulty in swallowing of several years' duration, with increasing difficulty during the preceding year. He could swallow liquids and finely prepared solids without any pain. He stated that food taken the night before was partially retained in the pharynx and some particles were expelled the following morning. Very often deglutition was accompanied by a choking sensation. He had lost considerable weight, but always had been under weight. During the World War he had been shot in the abdomen. In 1935 he had been discharged from a local hospital with a diagnosis of cholecystitis, hyperacidity, and arteriosclerosis.

Examination: The patient was of slender build and emaciated, was 5 feet, 10 inches tall (1.78 M.), and weighed 97½ pounds (44.35 Kg.). He had a long, thin neck without palpable lymph glands. Behind the thyroid cartilage on the right side, projecting forward on the body of the fifth vertebra, a firm knob could be palpated. The protuberance moved with the spine on rotating the head.

On laryngeal examination the tip of the right arytenoid cartilage appeared to be pushed forward into the larynx, and the motion of the right vocal cord was slightly impaired in abduction and adduction. The right pyriform sinus contained saliva.

Direct pharyngoscopy showed a cherry-sized protuberance pushing the posterior wall of the hypopharynx forward and giving the opening a triangular outline.

Under the fluoroscope the barium meal was seen to fill both valleculae, and entered the right pyriform sinus where it failed to pass but detoured into the esophagus through the left pyriform sinus.

The x-ray report by Dr. S. Brown was as follows: "The upper surface of the body of the fifth cervical vertebra shows an irregular

Fig. 1. Showing an osteochondroma arising from the bodies of the fifth and sixth cervical vertebræ, encroaching upon the esophagus. The bodies of the sixth and seventh vertebræ are fused.

Fig. 2. Section of tumor showing new cartilage and bone formation.

outline. From the front of the body there arises an exostosis which unites with another one arising from the body of the sixth cervical vertebra. The bodies of the sixth and seventh cervical vestebræ are fused together." (Fig. 1.)

The diagnosis was exostosis of the cervical spine compressing the hypopharynx and esophagus.

Operation: Oct. 5, 1937—Under paravertebral novocain block anesthesia on the right side and skin infiltration along the anterior border of the sternocleidomastoid muscle, a four-inch incision was made along the anterior border of the muscle. The carotid sheath was exposed and retracted laterally and, without encountering any important blood vessels, the incision was deepened by blunt dissection. The exostosis on the spinal column was exposed by separating the pharynx and the upper end of the esophagus from the prevertebral fascia. The upper end of the esophagus, together with the larynx, was then retracted to the left away from the midline. The exostosis presented itself in the form of a hemispherical knob, and was about 2½ cm. in diameter and about 2 cm. in height. After splitting the anterior longitudinal ligament, the exostosis was removed with a heavy rongeur and chisel by Dr. Joseph Freiberg, whom I had requested to assist me on account of his skill as an orthopedic surgeon. The bone had a rather soft, cancellous structure.

For the first 48 hours after the operation the patient was nourished through a duodenal feeding tube introduced through the nose. Thereafter he took all his food by mouth, and soon was able to swallow solid foods without difficulty. He was given small doses of insulin to improve his appetite and nutrition (Dr. B. Klein), but his weight increased by only a few pounds. He was discharged from the hospital on the twenty-third postoperative day.

Histologic Study of the Bone Fragments showed the presence of new bone and cartilage with a sarcomatous (?) island. (Fig. 2.) The section and x-ray film was sent to the Bone Registry of the American College of Surgeons, and Dr. D. B. Phemister reported: "I believe this is a hypertrophic arthritis of the spine with the formation of an enormous bridge between two vertebræ. The cartilage is a part of the process of enchondral bone formation in the laying down of the big bony bridge."

The patient was re-examined on March 16, 1938. He stated that he could swallow all sorts of food without any effort, but that on rare occasions he regurgitated small particles of food. X-ray ex-

amination showed great improvement in the passage of the swallowed barium, which descended through both pyriform sinuses (better on the left). There was no recurrence of the tumor.

SUMMARY

A case is reported in which dysphagia was caused by an osteochondroma of the cervical spine compressing the esophagus. After surgical removal of the tumor, the patient made a complete recovery and was relieved of practically all of his symptoms. There are several similar cases reported in the literature, but surgical intervention was not employed in any of these cases.

707 RACE STREET.

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LXIII

CARCINOMA OF ESOPHAGUS WITH UNUSUAL CLINICAL MANIFESTATIONS: REPORT OF CASE*

PORTER P. VINSON, M.D.

AND

PAUL KIMMELSTEIL, M.D.

RICHMOND, VIRGINIA

Lesions in the esophagus usually reduce the lumen of the tube and interfere with the passage of food or liquid into the stomach. When the obstruction is the result of a new growth, difficulty in swallowing solid food is almost always an early symptom.

Respiratory symptoms may be associated with diseases in the esophagus and are produced in a variety of ways. The esophagus may be dilated greatly in cariospasm, and when the organ is filled with food, respiratory distress resulting from pressure may be a prominent symptom. In certain cases of this type the symptoms may suggest asthma. Regurgitation and aspiration of food are noted frequently in cardiospasm and will lead eventully to chronic infection in the tracheobronchial tree.

In all cases of complete obstruction of the esophagus, regardless of the type of lesion that is present, regurgitation of mucus is inevitable. When this occurs at night, it is associated with aspiration and will lead to chronic bronchiectasis.

Fistula from the esophagus into the trachea or bronchi may be of congenital origin, may result from rupture of a dicerticulum of the esophagus, from penetration of a foreign body, ulceration of a granulomatous lesion, or most frequently from extension and perforation of a carcinomatous growth arising in the esophagus. When a fistula is present, the usual symptom is strangulation following the swallowing of fluid.

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From the Departments of Bronchoscopy, Esophagoscopy and Gastroscopy, and Pathology, Medical College of Virginia.

Marked reduction in the lumen of the trachea rarely results from extension of carcinoma of the esophagus into the trachea and, therefore, obstructive dyspnea is not observed often in cases of malignant esophageal disease. Extension of metastasis from a growth originating in the esophagus may involve the nerve supply of the larynx with resultant paralysis of a vocal cord and the production of moderate dyspnea and hoarseness. Such a condition is usually identified without difficulty from the character of the symptoms and examination of the larynx.

The following case of carcinoma of the esophagus is unusual because of the absence of dysphagia, the presence of dyspnea of an obstructive type, the character of the lesion, and the uncertainty of the diagnosis until postmortem examination was made.

REPORT OF A CASE

CASE 1.—A white man, 58 years of age, was referred to Memorial Hospital by Dr. C. B. Courtney of Newport News, Virginia, on October 23, 1937, because of progressive dyspnea which had begun two months previously. There were no other symptoms except loss of weight and strength and loss of sleep due to respiratory distress.

The general physical and laboratory examinations revealed nothing abnormal other than slight emaciation, marked obstructive dyspnea, and some difficulty in phonation. Roentgenoscopic examination of the thorax disclosed moderate dilatation of the arch of the aorta, but there was no evidence of a tumor in the mediastinum.

The respiratory difficulty was thought to be due to an intratracheal tumor, but on bronchoscopic examination the larynx was found to be normal and the lower portion of the trachea compressed laterally, especially on the left side, with the lumen reduced to about one-fifth normal size. There was no evidence of ulceration in the trachea, and tissue was not obtained for microscopic examination. Although the patient had not noted dysphagia and the lateral compression of the trachea was not considered to be the result of a lesion in the esophagus, roentgenoscopic study of the esophagus by Dr. F. B. Mandeville revealed an irregular filling defect in the middle portion, which was diagnosed carcinoma.

Esophagoscopy showed displacement of the esophagus by a mass located anteriorly. Each time that an effort was made to introduce the esophagoscope beyond this point the extrinsic tumor was pushed against the trachea and increased the dyspnea so that the tube had to be partly withdrawn. The examination, accordingly, had to be discontinued without visualization of a lesion in the esophagus.

The patient gradually failed and died November 28, 1937. Postmortem examination disclosed extensive flat carcinoma of the middle and lower thirds of the esophagus, 11 cm. in length, with little or no reduction in the lumen of the tube. The growth did not involve the entire thickness of the wall of the esophagus, but was practically limited to the mucous membrane except for an area 1 cm. in length in the center of the tumor, where infiltration of the submucosa had taken place. The muscular coat of the esophagus was not invaded at any point. Well above the tumor was a metastatic mediastinal mass which pressed on the left wall of the trachea, producing not only narrowing by direct pressure, but also an S-shaped distortion of the organ, which added to the reduction in the size of the tracheal lumen.

SUMMARY

A case of carcinoma of the esophagus is presented, in which all symptoms were the result of a metastatic mass in the mediastinum that produced tracheal obstruction. The only antemortem evidence of the presence of a lesion in the esophagus was obtained by roentgenoscopic examination. Esophagoscopic visualization of the tumor in the esophagus was not possible because of the character and location of the associated metastatic tumor.

300 MEDICAL ARTS BUILDING.

LXIV

SOME EXPERIMENTAL STUDIES OF PERORAL CATHETERIZATION OF THE COMMON BILE DUCT*

CHARLES J. IMPERATORI, M.D.

NEW YORK

In order to catheterize the common bile duct perorally, the duodenoscope devised by the author with the collaboration of Dr. E. C. Hanssen of New York is used. To Mr. F. C. Wappler of the American Cystoscope Company, all credit must be given for the original instrument through which the duodenum was first visualized perorally, and for the recent improvement that permits of catheterization of the common opening (bile papilla) of the common bile duct and the duct of Wirsung. The use of water, instead of air, in the ballooning of the duodenum has added a material factor of safety and definitely improved the visibility. The visualization of the duodenum is difficult mainly because of the similarity of structure of the interior of the viscus. By resorting to measurements and markings on the duodenoscope and using the pyloris as a guide, the structures of the duodenum about which we are concerned, may be searched for and found. Repeated practice is required in order to acquire any degree of exactitude and expedition in recognizing the anatomical structures searched for. The most important one is the longitudinal fold of the duodenum, at the lower part of which is the bile papilla. This slight elevation contains the orifices of the ductus communis choledochus and pancreatic duct. The slight elevation of mucous membrane caused by the union of the two ducts is called the diverticulum of Vater, or the papilla major. This longitudinal fold crosses the valvulæ conniventes and is the only permanent longitudinal fold in the small intestine. Somewhere nearer the stomach is a wart-like elevation, the opening of the accessory duct (santorini) of the pancreas. This is called the papilla of Vater, or the papilla minor.

The improved duodenoscope is the instrument used in the catheterization of the bile papilla. After introduction of the duodenoscope on the guided thread, and the beak has passed 7 to 9 cm. beyond the

*Presented before the Meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

pyloris, the papilla is sought. It is found on the posterior wall of the duodenum slightly mesially. The cannula is deflected sufficiently to enter the opening in the papilla. By the retrospective vision, the tip of the cannula can be guided into the opening. Aspiration of the duct contents is then done or a radiopaque media such as sodium iodide 12 per cent is injected. About 10 cm. is sufficient to outline the ramification of the duct of Wirsung, and by an added injection of 10 cc., the intrahepatic ducts are entered, using possibly 20 cc. in all. Roentgenograms are then taken.

As yet, this procedure has not been used on the living. First, because the required skill has not been attained to enter the opening of the bile papilla. Secondly, because of the possible injury to the cells of the pancreas or liver by the injection of the sodium iodide.

A suitable nonirritating radiopaque substance must be found and used. Thought must be given to the possible production of jaundice or hypoglycemia, by direct irritation of the chemical substance used as the radiopaque.

PRECIS OF THE EXPERIMENT

1. The study of the bile and pancreatic juice without contamination of the salivary esophageal, gastric and duodenal secretions.

2. The study of the patency of the cystic and common bile duct; also the ducts of Wirsung.

3. Indirect visualization of structures adjacent to the ducts by roentgenograms.

Further experimentations and studies are required before this method or any similar one can be used generally.

108 East 38th Street.

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LXV

CERTAIN CONSIDERATIONS ON DYSPHAGIA ASSOCIATED WITH ANEMIA*

LEIGHTON JOHNSON, M.D.

BOSTON

Practically all investigators of neoplasms agree that chronic irritation is of etiological significance in carcinomata of the mouth and pharynx.

The excessive use of tobacco and alcohol are the most common sources of irritation to the oral and pharyngeal mucosa, although dental caries is always a considerable factor. Because the male particularly has indulged excessively in the use of tobacco and alcohol, the incidence of malignancy is so much greater in this sex than in the female. In fact, the presence of cancer in the female has been often used as an argument against the validity of Virchow's theory of irritation.

While the theory of irritation holds for the male, carcinomata of the mouth, pharynx and esophagus in women has recently been observed to be etiologically dependent, not upon irritation but rather upon an achlorhydric or hypochromic anemia with its resultant local changes in the mucosa of the pharynx, mouth and esophagus.

Billroth was of the opinion that carcinoma always occurred in pathologically altered tissue, and it is not remarkable that he has so many supporters of his theory when one considers the predisposition of scar formations, atrophies, chronic inflammatory processes, such as tuberculosis and syphilis, to terminate malignantly. It is the atrophic changes in which we are most interested.

Simple achlorhydric anemia was first described by Faber in 1909. Anemia in women associated with dysphagia was observed by Plummer in 1914. In 1922, Vinson augmented Plummer's findings, and since then the syndrome of dysphagia associated with anemia has been called Plummer-Vinson syndrome. Ahlbom of Sweden believes

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that achlorhydric anemia and Plummer-Vinson syndrome are one and the same disease.

The most important symptoms of Plummer-Vinson syndrome are a hypochromic anemia, dysphagia, atrophic changes of the lips and oral mucosa, a smooth tongue, cracking or fissures of the corners of the mouth, early loss of the teeth, frequent changes in the nails (concave or spoon-shaped in appearance) and splenomegaly.

Wassink, in a lecture before the International Cancer Congress in Brussells in 1936, and Ahlbom of the Radium Institute of Stockholm in articles written both in 1936 and 1937, stated their convictions that this syndrome presented a specific predisposing factor in the etiology of oral, pharyngeal and esophageal cancer in women.

The Plummer-Vinson syndrome puts in its appearance usually from fifteen to twenty years of age. It may appear later but never comes after fifty. Malignant changes then appearing in the fourth, fifth or sixth decade of life may have had their inception with a simple hypochromic anemia at the age of puberty. Many cases of Plummer-Vinson syndrome are missed because of a faulty history of dysphagia. The dysphagia is sometimes a minor symptom. The patient feels that he has a small opening into his esophagus and will not admit of any difficulty in swallowing except under the most insistent questioning and, again, many patients having difficulty in swallowing and probably falling into the group of Plummer-Vinson are not checked for anemia and atrophic changes to the oral mucosa, but are classified as neurotics and as having globus hystericus. Jackson reports that, out of one hundred and ten cases of cancer of the esophagus, eighty-seven were at some time diagnosed as neurotics.

The dysphagia probably is caused by the atrophy of the mucosa in the hypopharynx and an atony of the muscles of deglutition. It is thought, however, by some observers to be due to spasm. In the severe cases of dysphagia, webs or bands are frequently seen in the upper esophagus.

The postcricoid region is the most common location for the carcinoma, and 90 per cent of all postcricoid carcinomata are in women.

Formerly, poorly fitting dental plates in edentulous women were blamed for the malignant changes, but as previously stated, the early loss of teeth is a symptom of Plummer-Vinson disease, and the syndrome or precancerous state is well established before the patient acquires artificial dentures.

Why women should constitute such an overwhelming majority of Plummer-Vinson patients is still somewhat speculative. Hypochromic anemia is nearly always confined to women, and Ahlbom ventures the etiological suggestion that the iron content of the blood is depleted by menstruation, pregnancy and lactation. This syndrome is usually found among women whose diet has a low iron content. Hence it is more common among the poorer classes whose diet is low on fruits and green vegetables. The Swedish peasants live on a diet which has a strikingly low iron content, and it is in Sweden that this syndrome is so prevalent.

The treatment of Plummer-Vinson syndrome is iron and careful and repeated endoscopic examinations of the hypopharynx and esophagus. If webs are present, they must be excised. In the absence of webs or bands, deglutition is often greatly improved by a simple diagnostic esophagoscopy. This improvement is further enhanced by the administration of proper doses of iron.

Any patient with anemia should be watched carefully and followed for a number of years. It is hoped that the early recognition of this state and its intelligent and persistent treatment may forestall the atrophic changes and their transition into a malignant state.

CONCLUSIONS

1. The irritation theory as a cause for cancer in men is tenable.

2. Cancer may appear on pathologically altered mucosa. The mucosa is altered in Plummer-Vinson syndrome.

3. Plummer-Vinson syndrome is a precancerous state.

4. Dysphagia with anemia is sometimes difficult to elicit.

5. The propaganda for cancer prevention should mention hypochromic anemia.

REPORT OF A CASE

CASE 1.—This is the history of a 54-year-old white woman referred to me from Aroostook County, Maine. Her chief complaint was difficulty in swallowing, which she said she had had for more than twenty-five years; it had been worse, however, for the last three months. She also stated that she had been anemic since childhood and had been treated for it most of her life. Because office examination revealed a frail, emaciated, edentulous woman with a smooth tongue, fissures at the corners of her mouth, obviously anemic and unable to swallow even liquids, it was felt that here, possibly, was a malignancy superimposed on a Plummer-Vinson syndrome, and she was sent to the Evans Memorial for study.

There they found her family, marital and social history negative. Under previous hospital entries, it was found that she entered a Boston hospital in 1931 and remained there fifteen days. While in the hospital, she had had her esophagus

dilated twice under general anesthesia. A hospital diagnosis was made of migraine exhaustion, general web of the esophagus, malnutrition, and irritable colon. The hospital history, however, stated three interesting and suggestive symptoms of Plummer-Vinson syndrome. First, "that she had been treated for anemia for years." Second, "that she had always had some difficulty in swallowing." Third, "that esophagoscopy revealed a web in the upper esophagus."

The past history as elicited at the Evans Memorial showed that she had never been well, always frail, anemic, and had difficulty in swallowing for more than twenty-five years. She had had much trouble with her teeth, the last of which were extracted in 1925.

The present complaint was that her dysphagia had been getting worse for the last three months and that now she could scarcely swallow her saliva. She had lost twenty-two pounds in the last four months; present weight eighty-eight pounds.

The physical examination was essentially negative except for her obvious anemia and a pale, smooth tongue.

The x-ray examination showed an obstruction just below the pyriform sinuses.

The endoscopic examination of the hypopharynx showed an extensive ulcerated postcricoid tumor. Biopsy was done and the condition diagnosed as epithelioma, grade III.

Because of her inability to swallow, a gastrostomy was performed. Following her gastrostomy she was able to take nourishment, and this, together with blood transfusions and other supportive measures, effected a moderate improvement in her general condition. She was then referred to the x-ray department for deep therapy. During the course of x-ray treatment she developed some edema of the larynx, requiring tracheotomy. She convalesced well from this operation and the x-ray therapy was continued.

In spite of a Type III (supposedly) radiosensitive tumor, and in spite of 5400 "r" directed to the affected area, there was little if any regression in the tumor size. She was eventually sent home to her family doctor for terminal care.

It is interesting to speculate on the possibile outcome of such a case if it were seen today in its incipiency.

15 Bay State Road.

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LXVI

A CONSIDERATION OF SOME OF THE CAUSATIVE FACTORS OF VEGETAL BRONCHITIS*

FREDERICK T. HILL, M.D.

WATERVILLE, MAINE

The object of this paper is to report a rather unusual case of organic foreign body and to attempt certain deductions which might be of some value in a consideration of vegetal bronchitis. This term was suggested by Jackson, Tucker and Clerf[1] in 1925, as covering all toxic reactions from aspiration of vegetal substances. This is thoroughly described in Jackson's[2] textbook and the clinical picture is familiar to all. A rapidly increasing toxemia leading to asphyxia, with cough, asthmatoid wheeze and the signs of bronchial obstruction, plus roentgen evidence of obstructive emphysema, or atelectasis, in a young child, immediately suggests the necessity of bronchoscopic examination. All authorities agree as to the imminent danger of early fatality, if not relieved. Jackson states that age is the most important general factor and that the severity varies inversely with the age. He also considered the question of a foreign protein reaction and of irritation from chemical substances in the inhaled foreign body.

In 1930, Heatley[3] reported a splendid piece of work based on studies of peanut bronchitis. He felt that the foreign protein theory was untenable, as roasted peanuts, in which the protein is insoluble, cause a severe bronchitis, while fat-free residue of raw peanuts, with protein little, if any denatured, produces no reaction. As a result of postmortem studies and animal experiments, he concluded that arachidic (peanut) bronchitis was due to irritation from free fatty acids found in peanut oil, in both the linolic and oleic fractions, and that infection played only a secondary role. Very slight concentration, 0.3 per cent, of free acid is sufficient to produce this condition.

As a result of these investigations there has been a natural tendency to consider the chemical or irritative factor of paramount importance with organic foreign bodies in the respiratory tract. At

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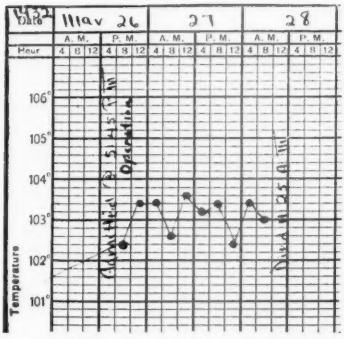

Chart 1. Case 1

times this may have resulted in disregarding other factors which might have been of equal, or even more importance, in certain cases.

REPORT OF CASES

CASE 1.—A male baby, age two years, was seen in consultation with the family physician. He had been in good health until two days before, when he began to choke and manifest respiratory difficulty. This had been considered and treated as "croup." There was no history of foreign body aspiration. The child was quite cyanotic and was breathing with great difficulty. Examination with a laryngeal mirror showed what appeared to be a foreign body below the vocal cords. The child was immediately rushed to the hospital, post-haste, with disregard of all traffic lights. On arrival he was taken at once to the operating room, where direct examination revealed marked subglottic edema and a large foreign body in the trachea. On removal this proved to be a large piece of celery stalk. The bronchi were now examined, with negative results, except for marked inflammatory reaction throughout. Tracheotomy was performed, leaving the bronchoscope in situ until the trachea was opened. Temperature on admission was 102 degrees. This ranged between 103 and 104 degrees from then on. (Chart 1.)

Despite all efforts to keep the airways clear there was continually recurring bronchial obstruction due to crust formation. These were removed by suction and by frequent lower bronchoscopy, but the baby expired 36 hours later.

Permission for postmortem examination could not be obtained, but careful bronchoscopy after death failed to reveal other foreign bodies.

This seems somewhat analogous to the case reported by Stiles,[4] where a piece of potato was found in the trachea. While this was removed eight hours after aspiration, the convalescence was quite stormy. Tracheotomy and subsequent frequent removals of crusts and plugs from the trachea and bronchi were required.

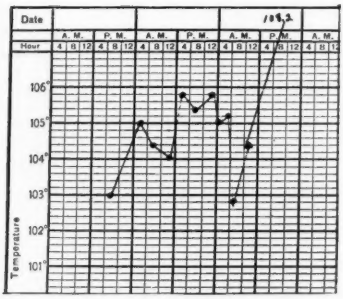

Chart 2

The rapidity and severity of the reaction might suggest a similarity with the aspiration of inorganic chemicals. In 1932, I[5] reported a case of lye burns of the larynx, trachea, bronchi and esophagus, in an 11-months-old baby, due to the aspiration of a washing powder containing sodium perborate, which forms sodium hydroxide in aqueous solution. This resulted in marked toxemia and death within 48 hours. (Chart 2.)

CASE 2.—Last year I had a similarly tragic experience with a baby 17 months old, who had aspirated a mixture of linseed oil and hydrochloride acid which had been left in a bottle on the floor. Breathing was noisy and apparently obstructed by mucus. Temperature 103 degrees. Bronchoscopy revealed large amounts of thick, stringy mucus in the larynx and trachea. The vocal cords appeared normal but the mucosa of the trachea and bronchi were beefy and red throughout. The esophagus was not involved. The mucus was cleaned out by suction. Roentgenogram showed a marked pneumonic involvement of both lungs. The following day there was increased difficulty in breathing and tracheotomy was performed. Breathing was easier after this, but the large amount of secretion necessitated the use of suction every five to ten minutes. Bacteriological examination of this showed pneumococci. There was marked crust formation which required frequent bronchoscopies to effect removal, most of these being done in the wee small hours of the morning. Normal saline was used in an endeavor to soften these crusts. Despite our best efforts to keep the airways clear, the baby died in eight days. Permission for postmortem was refused. (Chart 3.)

My first impression of the celery case was that it was a most violent toxic reaction, probably due to some chemical component of the inhaled foreign body. This was further enhanced by a conversation with one of the most prominent bronchoscopists in the country, in which he told of a similar tragic experience with celery as a foreign body. Later I learned of another fatal case due to celery.

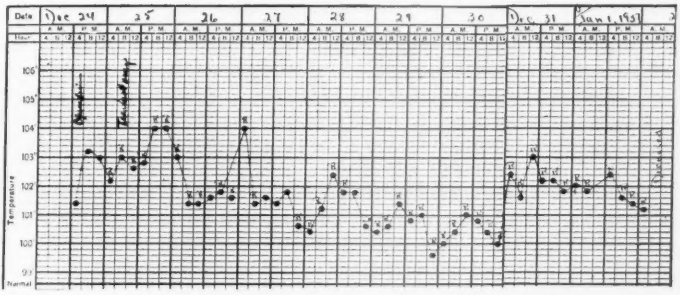

Chart 3. Case 2

Inquiries of a physician connected with the case revealed that a 3½-years-old child had choked on a piece of celery, dying 24 hours later. An emergency high tracheotomy had relieved the obstruction somewhat, but no bronchoscopic treatment was available and no attempt had been made to find and remove any foreign body. The physical findings and roentgenograms, I was informed, were typical of a marked pneumonic process involving both lungs equally.

Here apparently we were dealing with a substance which was much more rapidly toxic in the respiratory tract than the more usual vegetal invaders. In my case death ensued within 84 hours despite bronchoscopic removal, tracheotomy and as good after-care as we could give. In the case where the celery was not removed, death occurred in 24 hours, with both lungs markedly involved and simulating a severe pneumonia. This was a more rapidly severe process than usually encountered with peanuts, and we had become accustomed to consider the peanut as the most violently behaving foreign body.

I am indebted to Dr. George F. Parmenter,[6] Professor of Chemistry at Colby College, for the following information relative to the chemical components of celery. The edible portion of American celery is made up as follows:

Water	94.4%
Protein	1.4%
Fat	.1%
Carbohydrate	3.0%
Ash	1.1%

The inorganic substances in celery are the oxides of calcium, magnesium, potassium, sodium and phosphorus (phosphates) and chlorin and iron. The chief carbohydrate is mannital, while asparagin and tyrosin make up the protein components. The odor and flavor are due to essential oils, as glycerides. There are minute traces of terpene and sedanolic and sedanonic acid, and even lesser amounts of palmitic acid and phenolic substances.

Heatley found the chemical irritants in peanut oil, causing the bronchial reaction, to be the free fatty acids. However, as the total fat content of celery is only 0.1 per cent, and this is almost wholly in the form of glycerides, which are neutral esters, there would seem to be too little free fatty acid available to be of any significance. The only way this could be present would be by hydrolysis of the glycerides. This is dependent upon moisture, temperature and enzymes. Pinkerton[7] has shown that vegetable oils are unaffected by lipases present in the lung and therefore are not hydrolyzed. Consequently it seems fair to assume that chemically at least the problem is entirely different from that of the peanut, which has a comparatively high oil content, and that the rapid and severe reaction was due to other factors.

As Jackson pointed out, peanuts and similar substances which do not tend to swell, usually allow air to get past, and cause a trapping obstructive emphysema. Because of their smaller size these are more apt to lodge lower down in the bronchial tree. Depending upon the point of lodgement, at first a comparatively smaller pulmonary area is involved. This soon extends as the toxic chemical reaction from the free fatty acid is set up, until the whole lung is involved. Heatley observed postmortem involvement extending even to the other lung. The younger the child, the smaller are the airways in proportion to the foreign body, and the higher the probable point of lodgement; hence the greater initial lung involvement. This is probably one reason for the increased severity of this condition in younger children. However, there is a definite time element, the length of sojourn necessary for this chemical reaction to develop, which is quite out of proportion to our experience with celery. We have all had cases where early removal of a peanut foreign body has resulted in prompt recovery without any necessity of tracheotomy.

CASE 3.—A girl of five years aspirated a peanut kernel 24 hours before. She had fever and an asthmatoid wheeze. Bronchoscopy revealed a half a peanut kernel in the right main bronchus, which was removed. Temperature gradually came down to normal and the child was discharged from the hospital in five days. (Chart 4.)

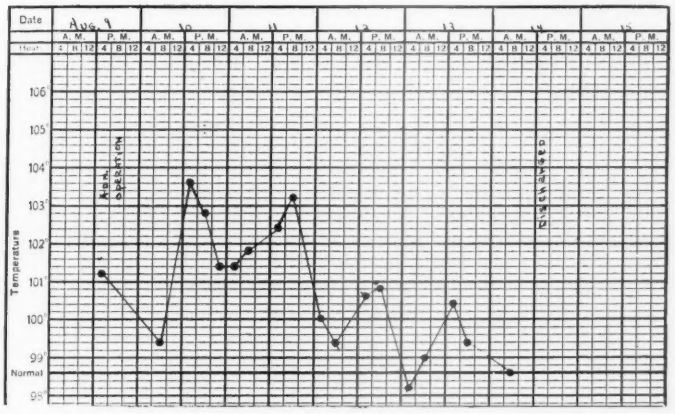

Chart 4. Case 3

Unfortunately the time interval between aspiration and bronchoscopy is usually longer, and consequently the problem more complicated. The following case offers a distinct contrast.

Case 4.—A baby, 2½ years old, in very poor condition, was seen 38 days after aspiration of a peanut. He had been extremely ill during this time. Roentgenogram and physical examination showed a complete obstruction to the bronchus to right inferior lobe. Bronchoscopy revealed the tracheal and bronchial mucosa markedly inflamed and foul smelling secretion coming from the right main bronchus. This was filled with easily bleeding granulations. After removing these with forceps, an irregular fragment of peanut was seen and removed. Five hours later tracheotomy was required. There was a large amount of secretion, which would spill over into the other lung, necessitating hurried bronchoscopic aspirations, but which finally resulted in the death of the baby two days later. Autopsy was not permitted, but a postmortem endoscopy was performed. No more foreign bodies were found but frank pus was seen coming from the right inferior lobe, indicating a case of lung abscess and drowned lung, secondary to a vegetal bronchitis. (Chart 5.)

Of course this case is somewhat extreme as far as the time interval is concerned, but it illustrates the usual end-result of a peanut foreign body in the bronchus for sufficient time for the chemical reaction to progress. On the other hand, Kernan,[8] in 1926, reported a series of cases of aspirated peanuts ranging from one month's duration to twenty-three years, all living, although with marked destruction of lung tissue.

Other vegetal substances may simulate, in part, the behavior of the peanut foreign body, yet cause little or no chemical reaction.

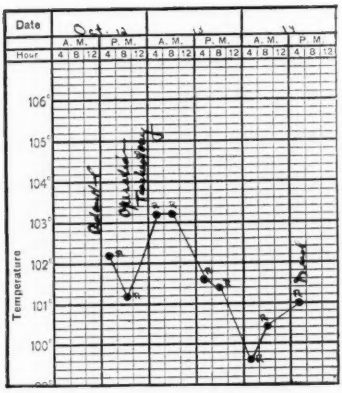

Chart 5. Case 4

CASE 5.—A baby, 18 months old, choked while eating a peeled apple and aspirated some of the pulp. There was considerable difficulty in breathing, and when seen six days later, there was a definite obstructive emphysema on the left. Bronchoscopic examination showed a reddened, inflamed mucosa and copious secretion in both main bronchi. This was removed with suction. No foreign body was found. This procedure was repeated in two days, with similar results. Following this, fluoroscopic examination showed the left diaphragm, previously fixed, moving normally with respiration. For a time it seemed that tracheotomy would be required but respirations improved until three days later, when another bronchoscopy was done because of an increase in obstruction. A large white cast, apparently membrane or slough, was found in the trachea and removed. Following this the temperature, which had ranged between 102 and 104 degrees, gradually came down to normal and the child was discharged from the hospital one week later.

While no definite foreign body was found, evidently the apple pulp was removed by suction together with the thick, tenacious secretion and the emphysema relieved. Apple pulp has no more potential chemical irritants than has celery. Undoubtedly the factor here was mainly mechanical.

Reverting again to a consideration of celery, it is probable that any portion of the stalk inhaled would be much larger than the usual half of a peanut, or fragment, which is aspirated, and therefore is more analogous to the bean. At least this seems to be the case, as far as our limited experience goes. Jackson considers that beans are the most often fatal of all foreign bodies. Acting as ball valves, they cause atelectasis. As they swell, due to the absorption of

moisture, they exert a tremendous pressure and are fitted tightly into the surrounding mucosa. With the absorption of the residual air the atelectasis increases. Beans, because of this property, as well as their comparatively larger size, are prone to lodge rather near the bifurcation, resulting in an initial blocking and involvement of a large pulmonary area.

Case 6.—A boy, four years old, was seen the day following the aspiration of a large kidney bean. He had had considerable respiratory difficulty during the night. This had increased and he had been quite cyanotic when taken to St. Mary's Hospital in Lewiston in the morning. He was referred to me, and during the journey, a matter of fifty miles, his breathing became much easier. When he arrived there was no obstructive dyspnea, but examination showed no air passing into the right lung. Temperature 102 degrees. Bronchoscopy revealed considerable glottic and subglottic swelling, so that only a 4 mm. bronchoscope could be passed. Just below the bifurcation, in the right bronchus, there was a large foreign body, tightly impacted and completely filling the lumen. Because of the glottic swelling and the size of the foreign body, I feared stripping off at the vocal cords and the sucking down of the bean into the left (functioning) bronchus. Consequently I postponed any attempt at removal and performed a low tracheotomy. The following morning a bean, 2 cm. in length, was easily removed by lower bronchoscopy. Following this he made a gradual convalescence, although it required eleven days to decannulize him. My feeling is that the bean had been in the trachea until the morning of his admission to the hospital, when it was sucked down into the right bronchus. This would account for the sudden improvement in his breathing on the trip to the hospital. The presence of the bean, moving up and down in the trachea, prior to this, would explain the marked tracheal and subglottic reaction, as moveable foreign bodies in the trachea produce violent symptoms. The bean has rather a thick covering, and, as this was intact, it is doubtful if there was any irritative chemical factor in this case. (Chart 6.)

As before mentioned, this seems more analogous to the celery foreign body. The size and contour of celery stalk is such that it remains a tracheal foreign body and might nearly occlude the lumen in a small infant. It would not move up and down, as least as freely as a bean, but, from its size alone, could provoke a very severe reaction and result in marked obstruction, affecting the whole pulmonary tract. This would seem sufficient to account for the rapid and severe reaction without the necessity of chemical irritation from free acids.

So, while keeping in mind the importance of the chemical irritative factor with substances such as peanuts, which have free fatty acids in their composition, we must attribute a large part, at least, of the bronchial reaction with certain other organic substances to other factors. The mechanical factor must be of great importance, especially with the larger substances which tend to lodge higher up. Inorganic foreign bodies, if properly lodged, may often simulate quite the same picture.

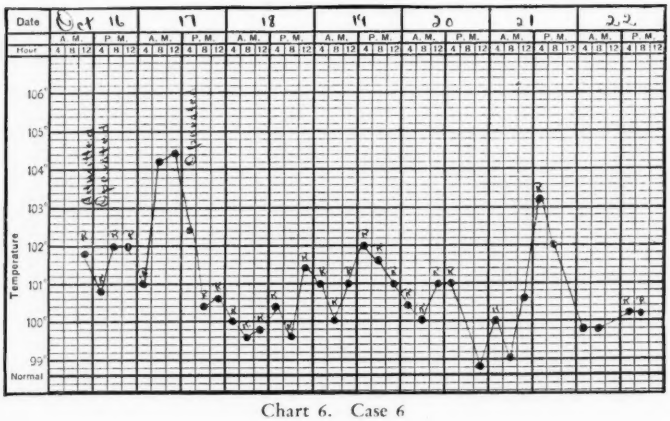

Chart 6. Case 6

CASE 7.—A boy, age 12, had been previously treated for pneumonia in another hospital. Temperature averaged 104 degrees. Finally a roentgenogram was taken which showed an upholstery tack, point uppermost, blocking the right main bronchus. The shaft was bent and the point imbedded in the wall of the bronchus. Removal released considerable pus from below. This was followed by uneventful recovery.

Substitute a younger age and consequent smaller airway, and add the increased pressure due to swelling of an organic substance, and we might have quite a similar picture, except for the probable extension as time went on.

Duration of the foreign body in the respiratory tract is of course most important in regard to the ensuing pathology and to the prognosis.

CONCLUSIONS

1. The aspiration of organic foreign bodies into the pulmonary tract results in a severe reaction known as vegetal bronchitis. There are several factors concerned in the production of this condition.

2. These factors are: the mechanical, the chemical, the duration or time element and the age of the patient. These factors may vary in importance, depending upon the kind of organic foreign body.

3. The age of the patient is the one most constant factor. The severity of the condition varies inversely with the age. This is due, at least in part, to the proportion between the size of the foreign body

and of the airways of the patient. The higher the point of lodgement, due to the smaller lumen of the younger child, the greater the area of initial pulmonary involvement.

4. The duration or time element varies, depending upon the nature and location of the foreign body. A high mechanical lodgement may produce a more rapid untoward result than a chemical irritation lower down where it is necessary for the chemical reaction to develop or extend.

5. The chemical factor is of extreme importance in those substances which have or may produce free fatty acids, as shown by Heatley. These resemble the picture produced by inorganic chemicals, except for being less rapid and severe in their action.

6. The mechanical factor is of importance especially with larger substances, which may be chemically inert. The reaction in these cases may be more rapid and severe than where the chemical factor is paramount.

7. Celery stalk as a foreign body may be rapidly fatal in its action, largely through this mechanical factor.

PROFESSIONAL BUILDING.

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LXVII

SUCTION, BATTERY AND OXYGEN UNIT FOR THE BRONCHOSCOPIC OPERATING ROOM*

PAUL H. HOLINGER, M.D.

AND

ALBERT H. ANDREWS, JR., M.D.

CHICAGO

This apparatus was designed to combine into a small efficient unit the double suction pump, a quickly available and easily controlled oxygen supply, and the battery boxes and table which are kept to the left of the operator in the bronchoscopic operating room. The unit consists of a cabinet 31 inches high, 24 inches long, and 16 inches wide, in which the suction pumps and motor are mounted. A rubber and spring mounting makes the pump practically silent in operation. The suction leads are carried to two vacuum bottles on the end of the cabinet nearest the operator. The motor has one-sixth horsepower and produces 26 inches of water vacuum.

The top of the cabinet serves as a table for two regular three-circuit Jackson battery boxes. These are kept in position by metal strips fastened to the cabinet top.

Two size D (80 gallon) oxygen-carbon dioxide tanks are placed horizontally beneath the cabinet on special racks. A pressure gauge registers the amount of gas in each cylinder. A flow-meter is next in series, to permit accurate regulation of the liters per minute which the patient receives. The gas then passes through a wash bottle and a trap bottle. Rubber tubing from this is kept in the sterile drapes and may be attached directly to the bronchoscope in an emergency. The drapes hang from the open tops of the battery boxes and cover the entire apparatus.

The door of the cabinet opens on the side away from the operating table. On the side next to the table, a rod extends six inches

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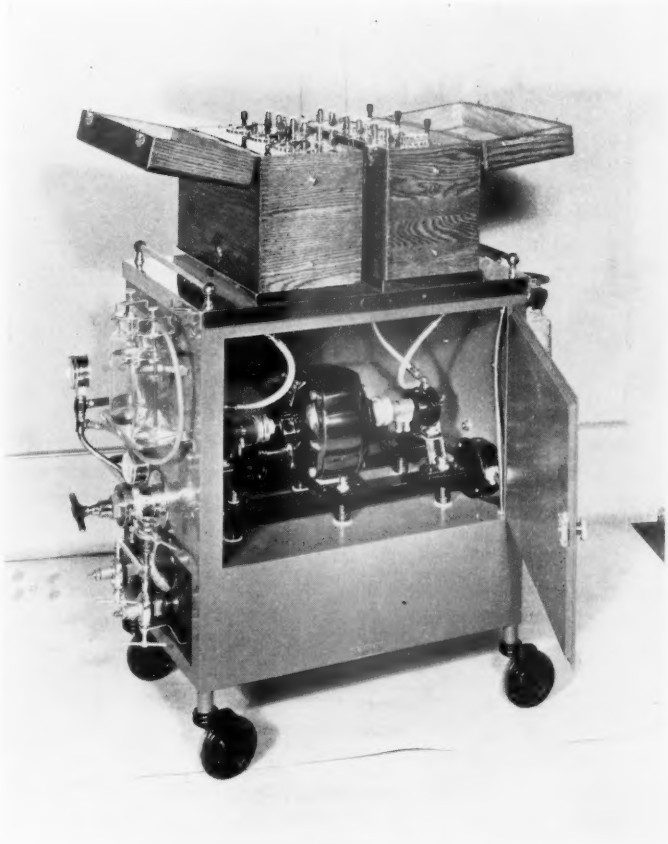
*Presented before the Meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

From the Departments of Bronchoscopy, St. Luke's Hospital, Children's Memorial Hospital, and the Research and Educational Hospital of the University of Illinois, Chicago.



Suction, Battery and Oxygen Unit.

from the cabinet and is draped to form a receptacle for the used light cords and suction tubes. The switch for the pump is of the mercury type, and is located so that it can be conveniently operated through the sterile drapings.

This unit combines three rather bulky pieces of apparatus into one which is easily movable, and keeps the necessary suction, compressed air, oxygen and light cords always available and within easy reach of the endoscopist.

707 FULLERTON AVE.

LXVIII

THE TREATMENT OF TUBERCULOUS TRACHEO-BRONCHITIS*

F. W. Davison, M.D.

Danville, Pennsylvania

The pathology and symptomatology of tuberculous tracheobronchitis have been well described in the recent literature. Therefore, I shall confine my remarks to the observations made on our own cases and to the ideas evolved from those observations.

Our first patient with this type of lesion was admitted to the Geisinger Hospital in October, 1935. She was very dyspneic and had all the signs of marked tracheal obstruction. Prior to her admission she had been treated for asthma. Bronchoscopy showed the tracheal obstruction to be due to marked submucous infiltration of the tracheal wall beginning 3 cm. above the carina extending downward to involve the circumference of the right main bronchus. The lumen of the trachea was not more than 4 mm. and the airway was further narrowed by considerable quantities of tenacious, grey mucopus. This exudate contained many tubercle bacilli. A tracheotomy was done and she was kept alive for nineteen months by means of a long, cane-shaped tracheotomy tube. Cicatricial stenosis of the right main bronchus eventually developed, producing retention of pus in the right bronchus and its tributaries. During this stage of her illness the usual signs of sepsis could be temporarily relieved by mechanical dilatation of the stricture and aspiration of the large quantity of pus which accumulated distal to the stricture. She died eventually because the left main bronchus became obstructed by tuberculous granulation tissue. The course of this patient's illness is similar to that of many cases described in the literature aside from the fact that tracheal obstruction in this case was temporarily relieved by the use of a cane-shaped tracheotomy tube. This case report illustrates clearly what happens to many of the patients who have tuberculous tracheobronchial lesions that are not treated. At that

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*Presented before the Meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

From the Department of Otolaryngology and Bronchoscopy, Geisinger Memorial Hospital, Danville, Pennsylvania.

time we did not have any means at our disposal for stopping the spread of these endobronchial lesions.

Our second patient was admitted for treatment in August, 1936. This patient, as well as the others described in this series, was referred by Drs. Devitt and Packard of Devitt's Camp, Pennsylvania. Despite the fact that her general condition was good and that good collapse of the right upper lobe had been obtained by pneumothorax, the sputum continued to be positive and a wheeze had been constantly present for the preceding five months. Bronchoscopy revealed an area of submucous infiltration in the right lateral wall at the bottom of the trachea, which merged into an area of ulceration surrounding the orifice of the right upper lobe bronchus. This area was covered with tough, yellow, adherent, fibrinous exudate which was the source of the loud wheeze. About that time we heard of the work of Drs. Warren and Hammond at the Herman Kiefer Hospital in Detroit. Because this patient had failed to show any improvement despite pneumothorax and strict antituberculosis regime, we decided to use electrocoagulation therapy. The ulcer healed completely in six months after a total of twelve treatments. Cicatricial stenosis of the right upper lobe bronchus was the final result, but this was not harmful because no purulent secretions were trapped distal to the stenosis. This patient is well and symptom free at the present time.

Our third patient had an ulcer 4 mm. in diameter on the posterior wall of the left lower lobe bronchus just below the orifice of the left upper lobe bronchus. This ulcer healed in four months after a total of eight treatments.

The fourth patient had a persistent wheeze in 1935, which disappeared following a total left thorocoplasty. Despite the fact that apparently efficient collapse had been obtained by phrenic interruption and thorocoplasty, the sputum remained strongly positive. Bronchoscopy showed marked infiltration of the submucosa of the left main bronchus with ulceration of the left lateral wall. There was marked stenosis of the left main bronchus at the time of the first bronchoscopy. The ulcerated area healed completely after a total of fourteen treatments given over a period of 10 months. The ulcer in this case was reactivated and healing was delayed by attempted mechanical dilatation of the strictured left main bronchus. The sputum has remained negative up to the present time.

The fifth patient was extremely emaciated and in very poor physical condition. She had ulceration around the orifice of the right

upper lobe bronchus and ulceration around the orifice of the right middle lobe bronchus, which spread outward onto the anterior wall of the right stem bronchus. She was given a total of twelve treatments over a period of eight months. The right middle lobe bronchus remained persistently closed by granulations. Tuberculous empyema developed on the right side, probably due to rupture outward of purulent exudate trapped in the middle lobe. Despite her extremely poor physical condition and almost constant fever, the bronchial lesions regressed in size during the period of treatment. Treatments were discontinued because of her hopeless general condition.

The sixth patient had moderate infiltration in her right upper lobe and an ulcer around the orifice of the right upper lobe bronchus which spread upward on the right-hand wall of the trachea for a distance of three-quarters of an inch. This ulcer healed completely after a total of fifteen treatments given over a period of nine months.

The seventh patient had ulceration surrounding the left upper lobe orifice which extended downward for a short distance on the left lateral wall of the lower lobe bronchus. The left lower lobe bronchus was completely occluded by caseous debris. The ulcerated areas healed in three months after a total of eight treatments. Her sputum has been negative for the past five months.

The eighth patient had ulceration around the orifice of the right upper lobe bronchus which extended up the lateral wall of the trachea for a distance of 2 cm. This ulcer was not so deep and was obviously of more recent origin than those in the cases previously cited. It healed in a period of four months after a total of seven treatments.

The ninth patient had the most extensive bronchial involvement of any of the cases previously reported, except the first. Her ulcer was of the diffuse type referred to by Drs. Warren, Hammond and Tuttle as a "granulating ulcer." It involved the entire circumference of the right main bronchus, the carina and the posterior wall of the trachea just above the carina and was beginning to spread down the mesial wall of the left main bronchus. This patient has had eight treatments up to the present time and is improving rapidly. Our observations of the gross appearance of these lesions as seen bronchoscopically correspond closely with the descriptions given by Warren, Hammond and Tuttle.[1]

There is no uniformity of opinion as to how often these lesions heal spontaneously, but the patients in this group showed no decrease

in symptoms due to their bronchial lesions until bronchoscopic treatments were begun.

The reports of Samson, Barnwell, Littig and Bugher[2] and of Bugher, Littig and Culp[3] indicate clearly that tuberculous patients who develop bronchial lesions have a poor prognosis.

From a review of our eight cases it is evident that patients with only minor parenchymal tuberculosis can develop bronchial lesions.

In the treatment of these lesions we have been using the Kernan electrode through either a 5, 6 or 7 mm. bronchoscope. We feel the current strength used should be of low intensity so as to minimize the possibility of producing necrosis of cartilage in the bronchial wall. Accurate standardization of current is probably not important, because the effectiveness of coagulation at a given point for a given current strength will vary, depending on the amount of mucus or other exudate between the electrode and the diseased tissues. We are at present using the Bovie electrosurgical unit and keep the current control at its lowest point unless a mass of tissue projecting into the bronchial lumen is to be destroyed. The length of time that the electrode is applied at any one point varies from one to five seconds. Generally speaking, we try to avoid producing coagulation deeper than 1 mm. Our purpose is not to attempt to destroy all of the tuberculous tissue present but to stimulate the reparative processes of nature.

Cauterization of tuberculous laryngeal lesions has been an accepted form of treatment for many years. Electrocoagulation probably has the same physiological effect as electrocauterization, namely, the production of a burn. This is beneficial probably because it induces hyperemia and hastens fibrosis. It should be kept clearly in mind that we are not endeavoring by means of these treatments to destroy all the tuberculous tissue present, but merely to hasten cicatrization of the lesion by stimulation of the natural reparative processes.

It seems apparent that these lesions spread by continuity along the submucosa. They seem to originate in most cases in the bronchus draining the affected lobe and spread outward into the next larger bronchus. Since we have available a means of inducing healing in small ulcers, it is important that the diagnosis be made early and treatment instituted before the ulcer has had time to involve the entire circumference of the major bronchi. It is obvious that there will be less scarring and contraction from healing of a small ulcer than there will be from healing of a large ulcer. If treatments are gently carried out and normal mucosa is not burned or traumatized, I do not think there will be any more cicatricial stenosis than would have

developed from spontaneous healing if that should occur. In our experience, mechanical dilatation of fibrous strictures has not been effective except for very temporary relief of sputum retention. Cicatricial stenosis of marked degree is still a serious problem for which lobectomy may eventually prove to be the solution. Cautery excision of these severe stenoses involves real danger of perforation of a bronchial wall.

SUMMARY

Eight patients with tuberculous tracheobronchial lesions have been treated over a period of twenty months by an electrocoagulation current of weak intensity. Treatments were given at intervals of two to four weeks. No harmful effects were noted as a result of bronchoscopic treatment, and in each case the tracheobronchial lesions showed a consistent tendency to heal. A total of eighty-four treatments were given. There were no laryngeal complications and no spread of the parenchymal lesions was observed.

Six of these patients are clinically well. One patient is still under treatment and is improving rapidly. Treatments were discontinued in the other case because of the patient's extreme debility and emaciation.

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Clinical Notes

LXIX

A VERY UNUSUAL CASE OF SUBCUTANEOUS EMPHYSEMA CAUSED BY A FOREIGN BODY*

Louis Daily, M.D.

Houston

In this journal in March, 1937, I reported "A Case of Subcutaneous Emphysema Caused by a Foreign Body." My case made the tenth reported in the literature, and the first where, on removing the foreign body, a prune seed, a tear in the bronchial wall was seen.

The case I am reporting has several unusual features.

REPORT OF A CASE

Case 1.—J. N., white, female, age 4 years. On the afternoon of December 19, 1937, her brother took her riding on the bars of his bicycle. They fell, and the child came into the house frightened and crying. The only visible injury she sustained was some scratches upon her legs. Four hours later the child developed heavy breathing, resembling asthma, and paroxysmal coughing spells, which kept up through the night. The family physician was called the next day and made a diagnosis of bronchial asthma. On December 22, in the afternoon, the mother noticed a marked swelling of the left side of the face, which extended to the neck and to the left side of the chest. In a few hours the swelling extended to the right side, first showing in the face and then the neck and chest. On the morning of December 23rd, the family physician, realizing the seriousness of the case, sent the child to the Jefferson Davis Hospital. She was admitted on the surgical service with a tentative diagnosis of ruptured lung caused by a fall.

Physical finding on admission: The child was well developed and well nourished; temperature 100, pulse 110; respiration 32 and very labored; moderate cyanosis of the face; the child was obviously

*Presented before the Jefferson Davis Hospital Medical Staff, February 17th, 1938, and the Harris County Medical Society, March 9th, 1938.

in respiratory distress. There was an emphysematous swelling anteriorly on the upper part of the chest and face, which was crepitant and tender on pressure. The heart was within normal limits on percussion, its action rapid, regular in rate and rhythm. The other organs were normal.

The blood picture: Hemoglobin, 75 per cent; red blood count, 3,850,000; white blood count, 15,000; polymorphonuclears, 70 per cent; lymphocytes, 19 per cent; monocytes, 3 per cent.

Report of x-ray findings: "There is a generalized subcutaneous emphysema of the chest. Lungs show no evidence of pathological changes. No fracture can be seen."

The child was placed in an oxygen tent and was given small doses of sedatives. The patient gradually improved, cyanosis almost disappeared and dyspnea was diminished. Temperature ranged between normal and one hundred degrees.

The oxygen was discontinued December 27th, 1937. The patient did well for two days. On December 29th, the dyspnea became worse. Cyanosis returned and the patient complained of pain in the hilus regions, and was returned to the oxygen tent. At this time the emphysema had almost completely disappeared. The air disappeared from the right side of the chest, neck and face and then from the left side of the chest, neck and face. At this time an x-ray showed infiltration of the lower left lobe.

I saw the child December 30th in the oxygen tent. She was breathing with great difficulty. There was marked retraction of the suprasternal notch, the epigastrium and also of the intercostal spaces. I had the child taken to the operating room for tracheotomy. On opening the trachea, she coughed up pus and a membrane of the same character we often encounter in diphtheria, but its cultures were negative. I intended to pass the bronchoscope following the tracheotomy and explore the tracheo-bronchial tree for a nonopaque foreign body. The child was doing very badly; she had coughing spells, the force of which tore loose some of the stitches and bled considerably. I decided to postpone further instrumentation until a future date when the child was in better shape. For the next few days the child was very sick. Her temperature ranged from 100 to 104 degrees. She was given oxygen constantly from an oxygen mask.

Physical examination on January 3rd, three days following tracheotomy, revealed areas of bronchial breathing in both basal

regions with fine showers of rales and small areas of dullness suggesting broncho-pneumonia.

The patient improved, and on January 5th she was able to breathe comfortably without oxygen for several hours at a time.

Physical examination revealed an absence of breath sounds in the lower basal region of the right side which was thought to be due to a pneumonia and consolidation.

The next day, January 6th, I bronchoscoped the child with no anesthesia except for a preliminary hypodermic of morphia 1/12 grain and atropine 1 400 grain. The temperature at the time of instrumentation was 102 degrees. A small area of granulation tissue about 5 mm. long and 1 mm. thick was encountered in the lateral wall between the rings at the junction of the trachea and left bronchus. I picked it up with forceps but was afraid to tear or bite it off and applied 10 per cent silver. Beyond this, there was nothing unusual except for mucus in the trachea and left bronchus, which was aspirated. On introducing the tube into the right main stem bronchus, I encountered a solid plug of mucus obstructing the lumen. I removed the plug by suction; in addition to the mucus, it contained numerous small black particles, one about one-sixth of an inch in diameter. It felt and looked like wood. Distal to the obstruction, the bronchus was filled with a thick, creamy pus, which was aspirated. I had to use dry sponges to wipe away the thick pus from the wall of the bronchus.

Dr. Coulter, hospital pathologist, sectioned the largest one of the specimens and sent a slide to the Texas A. & M. College. They corroborated in the diagnosis that it was wood. Following bronchoscopy, breath sounds could be heard all through the lower lobe right side. The respiration became easy. Oxygen was discontinued. The tracheotomy tube was removed January 8th, 1938. The temperature subsided gradually to a level between normal and 100 degrees. On January 16th, 1938, the temperature rose to 103 degrees. There was a decreased resonance to dullness over the right lower lobe region with friction rub and moist rales on auscultation.

Report of x-ray taken January 17th, 1938: "There is a large area of rather dense infiltration of the right base." A diagnosis of right lobar pneumonia was made. I suspected that the right bronchus was plugged up again with thick mucus.

On January 19th, 1938, with the same preliminaries as before, I bronchoscoped the child. The granulation tissue was still present but not as marked as before. The mucous membrane of the right

Photomicrograph of the specimen—wood. Actual diameter 1/6 inch.

bronchus was congested, swollen, the lumen narrowed but not plugged up except for mucus, which I aspirated. The temperature at the time of instrumentation was 105.4 degrees. Three days later the temperature came down to normal, the patient improved rapidly, the chest cleared, tracheotomy wound closed and the patient was discharged from the hospital January 27th, 1938, in good condition.

This case presents many interesting features. Evidently, in falling off the bicycle, the child aspirated a piece of wood which punctured the wall of the left bronchus at its junction with the trachea. Through this puncture air escaped into the tissues. The foreign body lodged in the right bronchus, where, with the mucous secretions, it formed a solid plug, which blocked drainage distal to the obstruction. The physical signs and x-ray examination pointed to pneumonia at the base of the lung. Tracheotomy saved her life.

Bronchoscopic removal of the foreign body and mucous plug from the bronchus drained the right lung and saved the child from a fatal, obstructive pneumonitis, and established the diagnosis, which

could not have been made otherwise. The second bronchoscopy was of great benefit to the child, even though there was no definite obstruction. The aspiration of the mucus and the application of the adrenalin and cocain gave better drainage. Following the treatment the child made a miraculous recovery in a few days.

I wonder if many cases of pneumonia in children are not due to an obstruction of a bronchus by a plug of mucus, and bronchoscopy might save many lives. It can be performed without general or local anesthesia and without ill effects even though the patient's temperature, as in this case, is as high as 105.4 degrees.

Would it not be a safe procedure to resort often to bronchoscopy in various types of pneumonia where the lungs are filled with secretions and the child is too weak to cough it up?

I believe that the procedure deserves a trial, and should be called to the attention of the pediatrists who have the lives of these children in their hands.

I want to express my thanks to Doctors Johnson and Lyle, the residents at the Jefferson Davis Hospital, for the skillful assistance they rendered in this case.

1117 MEDICAL ARTS BUILDING.

LXX

SEPTAL SUBMUCOUS OPERATION WITHOUT NASAL PACK

ALEXANDER C. HOWE, M.D.

BROOKLYN, N. Y.

Some 20 years ago Lothrop[1] of Boston devised and published a report of a mattress suturing of the mucous membranes together, that excluded the distressing post-operative intranasal pack. It was not adopted widely because he used a fan-shaped mattress suture, the center point of the radiating sutures being close behind the initial incision. This frequently caused a septal perforation. I[2] published shortly after an account of a staggered mattress suture that I have used to the exclusion of all packing except in cases where the mucous membrane has been torn, when a very limited pack was used. This procedure has largely surplanted packing in the Brooklyn Eye and Ear Hospital.

The Lothrop septal needle and hook are absolutely essential. The needle is about ⅝ of an inch long, at right angles to the shank or handle and is grooved on the upper surface. The eye is placed in the groove a short distance from the tip. The eye carries an 0 or 00 catgut suture. The handle about 2½ inches from needle is bent downward at 45° so the right hand does not obstruct the vision. The needle is bent to the operator's left. The hook has a harpoon-shaped tip, so that it can be inserted between the upper surface of the needle and the suture thereby hooking the suture and withdrawing it out of the opposite naris.

PROCEDURE

After completion of the submucous operation, a double wire self-retaining nasal speculum is inserted and by a 2 inch looped wire fastened to the spring connecting the two specula, it is strapped to the forehead at an angle that orients both nasal lumina. The suture, an 0 or 00 catgut, is threaded through the needle till it is midway between the ends of a long suture.

With the right hand the needle is carried into the left naris to the posterior rim of the removed septal window or hole and thrust through the mucous membranes into the right nasal lumen. A slender elevator pressed against the right membrane near the ex-

pected point of puncture helps the needle to perforate both membranes readily. With the left hand the hook is passed between the upper surface of the needle and the suture, the needle is then withdrawn into the left naris and out of the nose. The hook then draws the loop of suture it has engaged out of the right naris until the loose end of the suture is brought out of the right naris so there is a single strand of the suture on which the needle is threaded running into the left naris, through the septum and out the right naris.

The needle is again thrust through close to the upper rim of the septal window a little forward of the first insertion. The loop is grasped by the hook as before; the needle is withdrawn and handed to an assistant and then the loop is withdrawn out of the right naris enough so that the loose end of the suture on that side can be threaded through the loop and, by traction on sutures coming from both nares, the loop is drawn snugly around the suture in the right naris and the membranes between the two needle insertions are closely coapted. The needle is then inserted close to the lower rim of the septal window and the procedure repeated. In that way a staggered or saw-toothed mattress suture closely holds both mucous membranes together. For the last insertion of the needle, grasp the posterior border of the initial incision with forceps, draw it forward, insert the needle and pass it through the opposite mucous membrane as close to the cartilage as possible, thread the loop as usual and drawing snugly, tie the sutures taut below the septum. The sutures are absorbed in three or four days so do not need removal except in front of the initial incision.

Instruct the patient on reaching home or the hospital bed to sit up for six or eight hours and spray the nose freely every twenty minutes to two hours with a 50% solution of Epinephrine 1:1000. If the nose bleeds and there has been no laceration, holding the entire alæ of both sides closely and firmly against the septum for fifteen or twenty minutes will control the hemorrhage.

The result of this procedure is, there is less bleeding than with the usual pack; the patient usually breathes freely through both nostrils; there is never the intense reaction that always follows firm packing and that is always slow in subsiding; the patient has practically no pain and no recollection of a night of misery. Many patients even return to work the following day. It is practically never necessary later to pack the nose unless there has been a tear and then a small square of a Simpson sponge on both sides will care for that if the spray fails to control the bleeding.

If there has been a laceration, the suture can be carried from below the tear on the untorn side through the upper border of the tear on the opposite side and then from above through to the lower border of the tear, back above the insertion in the untorn membrane. This can be repeated in the longer tears. In through and through tears the opening can be closed in much the same way even if a horizontal incision near the nasal floor is necessary to freely coapt the edges of the tear.

40 South Oxford St.

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LXXI

A CASE OF STREPTOCOCCUS MENINGITIS WITH STREPTOCOCCEMIA—RECOVERY*

ALFRED LEWY, M.D.

CHICAGO

R. L., age 14, was admitted to the Eye and Ear Infirmary May 5th, 1937.

Diagnosis: Right otitis media, mastoiditis, subperiosteal abscess. Simple mastoidectomy was performed. The mastoid was mostly sclerotic. The sinus was located almost overlying the antrum. No sinus or dura was exposed. Four days postoperative, the temperature rose to 104.6 degrees, fifth day to 105 degrees, and subsided to normal on the eighth and thereafter convalescence was normal. No meningeal or other cause for the rise was discovered, but the interne reported that he had noticed a vertical nystagmus, which must have been transitory. Patient, who lived out of town, was discharged June 4th, 1937, with the ear dry and mastoid wound healed.

The patient was readmitted July 29th, 1937. There was headache of two weeks' duration, referred to the vertex and right side of the head, and blurring of vision. There had been vertigo, nausea and vomiting. Two days before admission there had been coma, but on admission this had cleared somewhat.

Examination: Slight discharge from the right ear; slight tenderness at the tip of the mastoid and old scar; neck rigidity; Kernig, Brudzinski, Babinski sikns positive; facial twitching, paresis of all right extraocular muscles and of the external rectus left. Blood: Hemoglobin 70; R.B.C. 4,060,000; W.B.C. 21,000 of which polymorphonuclear 87, lymphocytes 8, monocytes 4, eosinophiles 1. Urine: Albumin present, and a few granular casts, three red and five white cells per field. Spinal fluid, under pressure, cells 710 per cmm. hemolytic streptococci in smear and culture. Blood culture showed hemolytic streptococci.

July 29th, 1937, radical mastoid operation was performed, uncovering the dura in the middle and posterior fossæ and exposing the lateral sinus. No cells were found, but fibrous granulation tissue

*Read before the meeting of the Chicago Laryngological and Otological Society, April 4th, 1938.

was removed from the previous mastoid cavity. There was no thrombosis of the sinus. Dissection was continued along the posterior surface of the petrous bone, and the dura was retracted until the cisterna pontis lateralis was reached at the internal auditory meatus, where it was incised, and cloudy cerebrospinal fluid escaped under pressure. A gauze drain was inserted into the cisterna, the wound was lightly packed with iodoform gauze and left open for drainage.

Postoperative treatment: Sixty grains of sulfanilamide with ten grains of sodium bicarbonate were given as an initial dose and sixty grains per day by mouth daily thereafter, also two 5 cc. ampules prontosyl were administered intramuscularly. After four days the patient became very pale, with cyanosis of lips and fingernail beds, and the respirations were twelve per minute. Sulfanilamide was discontinued for three days and small doses of Fowler's solution were given three times a day. Transfusions of 250 cc. blood were given on the second, fifth and seventh days.

Course: The temperature remained over 100 degrees for 12 days after operation. Four successive cultures of the spinal fluid showed hemolytic streptococcus, the fifth being sterile. Three successive blood cultures also showed hemolytic streptococcus, the fourth being negative. The last positive culture of the spinal fluid was August 10th, and the cell count 1560. The urine was normal on the ninth day. The drains were removed on the fourteenth day. On that day the optic disks were still blurred, elevated ½ diopter and showed some flame shaped hemorrhages. Two days later patient complained of headache and vomited. The temperature became subnormal, pulse 92. Spinal puncture showed fluid under pressure and slightly cloudy. Sulfanilamide was continued and from that time on patient appeared convalescent. The diplopia continued for about a month, even after all evidence of eye muscle paresis had disappeared.

The patient was last seen in April, 1938. She has gained only two pounds since discharge from the hospital and looks anemic. Clinically nothing abnormal can be found except the absence of the left knee jerk. She is still under observation.

This patient coming into the hospital on the eleventh day after evidence of meningitis appeared, two days after onset of coma, with positive cultures of hemolytic streptococcus from the blood and spinal fluid, and with paresis of the eye muscles, certainly did not offer a favorable prognosis, and is presented to show what may be done today with properly directed treatment and appropriate surgery, when surgery is also indicated.

25 East Washington Street.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 7, 1938

THE PRESIDENT, DR. WALTER H. THEOBALD, IN THE CHAIR

Diverticulum of the Esophagus

LOUIS T. CURRY, M.D. (by invitation)

(Abstract)

Four cases are reported of pulsion type esophageal diverticula originating from the pharynx. The difficulties in treatment of the condition are emphasized. During the past twenty years there has been some controversy regarding the relative merits of the one-stage and the two-stage operations. The advocates of the latter claim that there is less danger of mediastinitis. The former group believes the patient is subjected to greater danger by two operations. There has also been considerable discussion about disposal of the sac. Some have twisted it and anchored it high up in the wound. If not too large it has been successfully invaginated into the esophagus without extirpation.

The symptoms of pharyngeal diverticulum are quite typical: dysphagia, regurgitation of fluids and later solid fluids; coughing and huskiness of voice are usual; an accumulation of saliva in the pharynx at night. The bulge of the sac may be visible in the neck. Often a fetid odor of the breath is noted. Boyce's sign can usually be elicited immediately after swallowing. A gurgling sound is produced by pressure of the hand on the side of the neck, due to pressed out swallowed air from the pouch. Diagnosis is made from the history and by roentgenologic study. Examination should be made with the esophagoscope to rule out malignancy.

The technique first described by Jackson and Gaub in 1915, as modified by Eliason, Tucker and Thigpen, is reviewed:

The author's summary is as follows:

1. Difficulties have been encountered in getting a thread swallowed which would pass into the stomach.

2. Repeated failures are possible in trying to find the subdiverticular esophageal opening.

3. It is often difficult to keep the esophagoscope in place while the surgeon removes the sac.

4. There have been complications, such as mediastinitis and pneumonia, following the first stage of the two-stage procedure.

5. In one of the cases reported, there was no sac, but a general enlarged upper end of the esophagus, which was fortunately not perforated in the search for a pouch.

DISCUSSION

DR. T. C. GALLOWAY: The laryngologist has one marked advantage over the general surgeon in operating on diverticula, in that he is accustomed to work in the depths with adequate illumination. It should be borne in mind that while a master of technique like Babcock may do a one-stage operation, and do it safely, for the average man, the two-stage is better.

The esophagus has no serosa, and its secretions are very infectious, so that unless one does a preliminary dissection to produce walling off and then a secondary operation above that area, there is great danger of descending infection.

Lahey has called attention to the fact that if the sac is fixed high up and the angulation removed, it prevents a filling up of the sac. This alone may suffice, and I think it is not always necessary to complete the procedure.

DR. PAUL H. HOLINGER: Discussion of esophageal diverticula always raises the question of single vs. two-stage operation. Babcock and Jackson have reported a series of seventy or more cases operated in one-stage without a fatality. Lahey reports a similar series operated in two stages with similar results. Therefore, in good hands, the procedures are equally safe. We prefer the single stage operation, with close co-operation and teamwork between surgeon and endoscopist. If the sac is thoroughly aspirated and brought into the operative field by the esophagoscope, its base can be demarcated by sutures on the outside which do not go through the esophageal mucosa. The esophagoscope is then retracted and guided into the subdiverticular esophagus over a previously swallowed string. With the sac thus isolated it can be packed off to prevent infected material

from passing down into the mediastinum, and amputated. The single stage operation is entirely satisfactory if the esophagoscope is used in this manner.

DR. JOSEPH C. BECK: My experience with this work was in the early days. I notice that Dr. Curry was apologizing for the fact that he did not report any deaths. The first case I did was a fatality. I dissected down below the clavicle and failed in properly walling off that area, which I think would have prevented mediastinitis and death. Regarding the single and double step operating, I have tried both. Of course, when one compares the work of Judd, Lahey and Babcock, it is numbers that really count. These men can talk about experience, and I think eight cases is all I have operated upon. Dr. Russell, at the Veteran's Hospital, did a case under my direction in which a double operation was done—twisting the sac and subsequently dissecting it out; the little technical point of refinding the sac in the second-stage operation is important in the use of these little brass metal clamps to have them attached to the sac high up in the neck.

I notice Dr. Curry spoke about Horner's syndrome coming from the recurrent laryngeal nerve. I wonder if he means that?

DR. SAMUEL SALINGER: I would like to congratulate Dr. Curry on his vast experience with pulsion diverticula. I cannot recall having seen more than four cases in my entire experience, and I can assure you I was unusually well pleased with myself in having made the diagnosis. So far as treatment is concerned, one would have to boast of a much larger experience to be able to speak with authority. Nevertheless, isolated reports, especially those which frankly expose the difficulties and possible complications are more than welcome. The procedure is more difficult than it looks, and for the occasional operator it would perhaps be best that the case be left alone or turned over to someone else to worry about. I am sure that a distinct advance was made in the surgical treatment of these cases when Jackson suggested the use of the esophagoscope as a guide, and there is no doubt in my mind that it has been the determining factor in many successfully treated cases.

DR. LOUIS CURRY (closing): I would like to thank the discussors for being so kind to me, and to thank Dr. Holinger for his suggestion for identifying the subdiverticulum part of the esophagus by suturing. The subdiverticular esophageal opening is in the anterior part of the sac and it is relatively harder to see. The sutures might be a great help in the identification.

In reply to Dr. Beck, I read that Horner's syndrome had been observed. I have never seen it.

Congenital Dermoid Cyst and Fistula of Dorsum of Nose

E. W. HAGENS, M.D.

(Author's abstract)

The present case was deemed sufficiently rare and interesting to warrant reporting. David F., aged 4, was admitted to the Cook County Hospital, April 17, 1937. The history showed that the boy was born with a pin-point sinus on the dorsum of the nose, from which, from time to time, blood, yellow material, hairs and watery fluid discharged. Two years previously the child bumped his forehead. A swelling and abscess developed and was incised. Since that time the incision had failed to heal completely. From time to time the forehead lesion would swell up and discharge and then the opening on the nose would become active.

The boy was first admitted on the surgical service where a tentative diagnosis of osteomyelitis of the frontal bone was made. Roentgenograms being negative, the neurosurgeon was called in. The diagnosis of "frontal fistula and cerebrospinal rhinorrhea" was made. As this was not confirmed, we were asked to see the boy, and made a diagnosis of congenital fistula and cyst of the nasal dorsum. At the first operation the fistulous sac and tract of the forehead were removed. Several months later the fistula on the nasal dorsum was attacked. At this operation it was found that a large portion of the sac was located below the level of the nasal bones and connected with the outer part through a small opening between the lower end of the nasal bones. The deeper part did not connect with the nasal passages, and was left in situ, but cauterized with phenol.

Both wounds healed satisfactorily, although the one on the forehead has become swollen and discharged on one occasion. A review of the literature shows only three reports in the American literature and about twenty-six altogether. Surgical removal is the treatment of choice. It is believed that early ectodermal inclusion in the frontonasal region best explains the etiology.

DISCUSSION

DR. FRANK J. NOVAK: This is an extremely interesting case because of its rarity, and yet we know that mixed tumors may occur any place in the body. I had occasion this afternoon to learn of a mixed tumor, parotid type, consisting of epithelium, true myxoma-

tous tissue and cartilage, located on the ankle of the patient—of all places to find a parotid tumor.

Dr. Francis Lederer: The American literature report of six cases has been taken as a criterion for Dr. Hagen's report. I believe that congenital cysts of the nasal dorsum must occur more frequently than this number would indicate. I have personally observed several cases of dermoids located in this typical area. The fact that this case presented interesting clinical phases is not uncommon, because frequently these cysts are subject to secondary infection. It is quite likely, similar to the fistulae that we find anterior to the auricle, that in the infected state these cysts should be mistaken for something else. Cysts of the nasal dorsum are not easily eradicated, because unless you succeed in thoroughly removing the lining of the sac, the lesion will recur. In Dr. Hagen's case, the invasion of the interior of the nose is an interesting feature. Possibly if he had to be concerned with an infant, he would have had to give considerable thought to cautious conservatism. While the eradication of the dermoid is important, cosmetics and the future development of the nasal structure must govern the attack upon the lesion.

In a case I observed in an infant girl, I had these facts in mind and did not completely surgically excise the cyst, but employed surgical diathermy to destroy the lining. There was an excellent postoperative result. Had Dr. Hagens been concerned with his child, as an infant, and had to consider the future development of that nose, would that have influenced his manner of treatment as it did mine?

His statement regarding but six cases in the American literature is more or less of a reflection on literature in general, making it unreliable from a statistical point of view.

Dr. E. W. Hagens (closing): I wish to thank Dr. Novak for his discussion, and in reply to Dr. Lederer, I wish to say there were twenty-six cases, not six—three in the American literature and twenty-six all told. I agree with you that there are probably more cases than are reported. The thing that struck me was the fact that this child was admitted on surgery, then went to neurosurgery, and it was only by accident that we found him. I thought we ought to call attention to that. Then the finding at operation was surprising, to find a large portion beneath the level of the nasal bone. I had supposed it was all on the top. I had him under ether anesthesia and did not use surgical diathermy, but used other caustics, and if he had a recurrence I would use surgical diathermy. I left the por-

tion below the level of the nasal bone and excised the above part, because I did not wish to break into the nasal passages. There is one case on record in which that was done. It took a long time to heal, with considerable scarring.

Osteoma of the Mastoid Process

STANTON A. FRIEDBERG, M.D. (by invitation)

(Author's abstract)

Osteoma of the mastoid process is a rather rare occurrence. A case was reported in which a bony protuberance behind the right ear was associated with a comparable growth on the hard palate. Symptomatology in this condition is often negligible and the growth may be removed with very little difficulty. The tumor under consideration was composed of compact bone. These tumors are usually benign and the differentiation between simple hyperplasia and true osteoma may be difficult.

Recent Advances in the Diagnosis and Treatment of Deafness

GEORGE E. SHAMBAUGH, JR., M.D.

(Paper published in this issue.)

DISCUSSION

DR. DANIEL B. HAYDEN: I see no real objections to the theory advanced by Dr. Shambaugh for the explanation of diplacusis. A condition resembling a serous labyrinthitis which he describes as existing in cases of diplacusis is acceptable, whether to believe the tectorial or basilar membrane to be the vibrating structure in tone perception.

DR. SHERMAN SHAPIRO: This paper is most interesting. The essayist referred to quinin. I recall another drug much more commonly used, which has been recently mentioned as a possible cause for some of these nerve deafness cases, particularly in this part of the country where quinin is not commonly used. I have reference to aspirin and the salicylates, which should be kept in mind by otologists.

Dr. Shambaugh has advanced a theory to account for high-tone or low-tone involvement in his cases which, as I understand it, is based on mechanical factors in the movements of the round and oval windows. This view may be correct. I noted, however, that in his reports there was mention of a catarrhal condition. Is it not

possible that the high tone loss in these patients was caused by absorption of toxins through the round window?

The theories of the essayist on diplacusis would, if proven, be a good argument for the Helmholtz ideas as to the hearing mechanism. I should like to know if some of the people tested by Dr. Shambaugh are musicians; otherwise it is difficult to understand how they could recognize a tuning fork sound as one or two tones higher or lower. Listening to the presentation and watching the audiograms, which show a variety of tuning fork findings in what Dr. Shambaugh terms nerve deafness, it occurs to me that the diagnosis is apparently made in some instances on the finding of diplacusis plus vertigo or other evidence of vestibular involvement if this is present. Under these circumstances, it would appear that we shall have to revise our ideas as to the value of tuning fork findings in the diagnosis of the type of deafness.

DR. HOWARD BALLENGER: I would like to ask Dr. Shambaugh if he excluded middle ear deafness, especially from occlusion of the eustachian tube, in all these cases, because I believe these results can be duplicated with inflation, with the possible exception of the diplacusis. I presume he has definitely excluded the middle ear deafness as an etiologic factor, but think it should be emphasized.

DR. AUSTIN A. HAYDEN: I have enjoyed the presentation of this paper very much, Dr. Shambaugh. I wonder whether the influence of quinin in the production of deafness has been properly evaluated? I say that because it is spoken of rather often as a rare thing, but I have seen within the past year six cases of quinin medication deafness; that is to say, that I thought were due to that, and they were not through the mother but through the individual, himself or herself. The facts were only elicited upon taking a very careful history, in which it was stated by the individual that large amount of quinin had been taken in youth, and that it was always accompanied by ringing in the ears.

When I lived at County Hospital as a medical student, there was a young woman there, the daughter of the chief engineer, who presented a striking idiosyncrasy to quinin, who, on just stepping into the drug store, experienced always the noise in both ears, and she became extremely hard of hearing. I think Dr. Taylor of Jacksonville, Florida, has done very well to bring this to closer attention than has been given it before, by his work. I was interested in hearing Dr. Shambaugh cite the very close analogy between that and iritis. There is sitting to my right a gentleman who can speak with

very considerable authority on the subject of recurrence of iritis, and he has not had, so far as I know, any labyrinthitis, but he certainly had up to the time the Shambaugh treatment was given him, namely, the removal of tonsils.

The salt-free diet in vertigo is certainly of service, as mentioned by Dr. Shambaugh. There was one thing he did not mention, that may not be well known. I saw a doctor, 74 years old, who suffered from vertigo but was very much improved by the withdrawal of salt from his diet, but was not absolutely free from vertigo until he had his allergy corrected, and the allergy was corrected by his going to Colorado. where he was perfectly free of symptoms, where the dry air was very beneficial to his hypertrophic rhinitis, and he is well there.

To conclude, I would like to call Dr. Shambaugh's attention to the fact that it would seem to me that the tests, and what Dr. Shapiro said I agree with, that unless one is well trained in music it is hard to tell whether a tone is one or two above or not, but if a binocular audiogram is made I have found the difference between the one ear and the other is very readily ascertained.

DR. W. A. SMILEY: In support of Dr. Shambaugh's diplacusis theory, I would like to relate the incident of an opera singer a few years ago, who had a secretory otitis media and a little nausea and vomiting. He has never been heard on the opera stage since, because he cannot tell whether he is on key or not. He hears the tones differently in each ear.

DR. E. J. BLONDER: I wish to ask Dr. Shambaugh if the nerve deafness improved in all of these patients with only the removal of the focus of infection? Was any other supplemental treatment given? Did the hearing in these patients vary in subsequent audiometer tests?

DR. G. HENRY MUNDT: It is a very interesting thing, I think, to have a man stand up before this Society and say something can be done for deafness. I congratulate Dr. Shambaugh for doing that. I do not know whether his comparing labyrinthitis to iritis has been propounded in the literature. I believe he is perfectly right in that theory. There is no question that in these people you can elicit a difference in tone perception if you try, if you are patient, and the patient does not need to be particularly well qualified in music for you to get that. I do not know whether management with salt-free diet and ammonium chloride works as well or not. I have tried both

and have removed foci of infection. I am inclined to believe that one gets better results following the method of management outlined by Dr. Shambaugh than by salt-free diet and ammonium chloride internally.

May I say that if you are using ammonium chloride and your patient cannot tolerate 22.5 grains three times a day in gelatine capsules, I give them enteric coated medication and carry them over until they lose that difficulty and then slip them back on the capsules. There has been some comment in the literature that it is not particularly efficacious, but it seems to work when I have used it. To go back to labyrinthitis and iritis, there is certainly a similarity in management of the two conditions.

Dr. Paul A. Campbell: Since I am Dr. Hayden's patient to whom he referred, and to whom I owe the preservation of my vision at 20/20 after several attacks of iritis, I may say that I do have a little nerve deafness. In my high tones above 5,000 vibrations there is a definite drop.

Dr. Francis Lederer: Dr. Shambaugh has suggested a number of points to which he has appended nomenclature and deductions which I believe to be entirely presumptuous. His interpretation of findings and results of therapy are most interesting, but inclusive. For example, the case of the patient with serum in the middle ear for twenty years. His deafness responded to an adenoidectomy. It is inconceivable that serum could be present in a middle ear without organization and a subsequent adhesive process developing. Then, too, the patient whose sudden vertigo cleared up one week after tonsillectomy. This was merely a response to the water depletion or "thirst cure" which Dr. Shambaugh unknowingly carried out, rather than removal of a focal source of infection. For the benefit of those of you who so thoroughly coincided with the essayist's attempt to parallel iritis and what he prefers to call "exudative labyrinthitis," let me remind you that "glaucoma" of the labyrinth had long been in use to express one possibility which existed in vertigo. The term "exudative" is one that needs clarification. It is possible that the term is ill-chosen. There is an entity which we have long recognized as toxic labyrinthitis. While Dr. Shambaugh's idea of edema or thickening of the basilar membrane may be correct, it is apparent that he takes a great deal for granted in interpreting a basic pathologic change, for which he has no actual proof in the cases in which he has observed, and tries to formulate a new entity of "exudative" labyrinthitis when the long recognized "toxic" labyrinthitis expresses equally as well his purpose.

The application of the term "low-tone" nerve deafness is interesting. It is possible that these are the same cases which we have been calling mixed or combined deafness. Unquestionably, the importance of diplacusis allows the examiner to conclude that the patient has a preponderance of his disturbance in the nerve.

In regard to the ingestion of quinin and its effect on the mother and the child, I wish to call attention to an observed fact, namely, that there exists a deafness which has been misinterpreted as being due to otosclerosis, or to quinin. I refer to the toxemias of pregnancy, which is again an example of toxic labyrinthitis. I have observed several cases of deafness which followed pernicious vomiting of pregnancy who have developed this so-called "low-tone" nerve deafness. These cases had previously been diagnosed otosclerosis purely on the fact that the hearing loss followed pregnancy. Are they not cases of "exudative labyrinthitis?" I believe that the term "exudative" is too loosely applied by the essayists without adequate proof.

Dr. George E. Shambaugh, Jr. (closing): I want to thank all the discussors. Dr. Shapiro brought up the point of non-musical people being unable to distinguish pitch. It is true that some are not able to tell the difference, but when diplacusis is present there is no hesitancy, they do detect a difference of tone. You have to be sure that intensity is the same. So many people confuse intensity with pitch. When you get the intensity the same the patient can tell the difference in the tone. It is significant, I think, that in the diseased ear the sound is unpleasant, harsh, non-musical, whereas in the good ear it is a pure, musical tone. The diagnosis of nerve deafness was not made by the finding of diplacusis, but by the usual tests, Rinne, Schwabach, and Weber—which was always referred to the good ear. In other words, all the usual tests for nerve deafness.

Dr. Ballenger wondered if middle ear deafness were excluded. All had both Politzer and catheter inflation when first seen, and in no case was hearing affected by inflation, nor was there any indication of fluid in the middle ear. Dr. Foster of Texas has a deafness that he is sure is from taking quinin. He says that many people in the south who have had quinin, have some degree of nerve deafness. Dr. Blonder asked whether any other treatment had been used. As I said, the tubes were inflated and an attempt was made to remove any local infection. The improvement was shown, and it did persist. I have had cases of long duration that did not get the hearing back. One patient had a fifteen year history. He still has diplacusis. I do

not expect to be able to improve his hearing any more than you can improve the vision in an old quiescent iritis.

Dr. Galloway asked if any of my patients died and willed me their ears—so far they have not. The conception of pathology is based entirely on the hypothesis—that you can only get a diplacusis from inflammatory change, perhaps edema or exudate. I used the term exudative labyrinthitis for these cases because they showed a diplacusis; some lesion of the vibrating membrane that I assumed was due to exudate.

Presentation of Radical Mastoidectomy with Reference to Status of Hearing

L. J. WALLNER, M.D.

(Abstract)

Six cases of chronic discharging ear, with a record of hearing before and after operation, were shown. Five of these were operated on by a modified Bondy type of operation, taking down the bridge and outer wall of the attic, removing the incus and leaving the drum membrane and malleus.

The question of what the hearing will be after radical operation is particularly important in bilateral cases, where the better ear is to be done, as in two of the cases cited, or where both are to be operated upon. In some the hearing may improve or remain stationary, but in the majority of cases an appreciable loss can be expected. Where better hearing results, it may be due in part to an improvement of a toxic neuritis of the eighth nerve from absorption from the process in the middle ear. Loss of hearing after a radical operation is ascribed to the removal of the drum membrane and ossicles, to dislocation of the stapes, or to fixation of the stapes in the scar resulting from granulations.

Skin grafts are said to preserve the hearing by lessening granulations. Two of these cases had no grafts, had long postoperative care, but now have normal hearing. The remarkable hearing in three of the cases seems to be due not to a functioning drum membrane and ossicular chain, as the incus was removed, but to the drum membrane protecting the region of the oval window from scarring. The case with profound loss of hearing after operation shows the stapes buried in a dense scar, which explains the poor hearing.

In none of the cases has there been recurrence of the cholesteatoma formation, and all have dry ears, with the exception that two have had brief periods of mucosal discharge during a head cold.

DISCUSSION

DR. JOSEPH C. BECK: I had the pleasure of seeing these patients before the meeting, and their ears were all nice and dry and just as described by the writer of the paper. I used to say to the boys training with me, "If you want a good practice later, do a lot of radicals early and wait, because the radically operated patient is yours forever, unless he gets disgusted and goes to someone else." The modified radical operation dates back to Winckler, but the Americans got the fever when Heath began to write about it. Heath, an Englishman, set the world on fire with cures by the Heath operation, not removing the bridge and having no facial nerve paralysis. We started it here in Chicago when the late Dr. William Ballenger and I did a number of operations, and this is what we found. In early cases it worked beautifully, but all the old chronic suppurating cases had to be reoperated, as the modified operation yielded no results.

The other operation mentioned—the removal of the external attic—brought a number of cures in the chronic suppurative cases, but the hearing was never as good as I have found it here tonight. When I asked them how the other ear was masked, they replied, "By sticking the finger in the ear." We know that this is not correct. I am just mentioning this as a possibility that the hearing might not be so good.

Now as to skin grafts—you are merely inviting debris in the ear—a foul smelling epithelium, which has to be removed every so often. You do not do skin grafting, you do epithelium grafting, and it is not well nourished. I think if the cases shown tonight continue to look as well as they do now, we should continue to do it. I would like to know how long it took these ears to heal As I told you, when the International Congress met in Boston, Heath had an opportunity to operate in a number of Eastern cities, such as Boston, New York and Philadelphia. When we obtained the reports on these cases they were very, very poor; in fact, they were terrible, as most of them had to be reoperated upon because they were not fit for such a procedure.

Then Bondy, or some of the later men, such as Blackwell, did this operation, and it was my pleasure to review some of these cases at the Infirmary in New York. They had been operated upon over a long period of time and were in excellent condition.

What he said about the production of membrane, as well as skin graft over the oval window may be true. In radicals that do

poorly in hearing, the scar formation knocks the hearing out or possibly in the thorough eradication a little low-grade labyrinthitis is started. When you have two ears that are suppurating, you are going to think before you do a radical on both ears. I have one girl that constantly reminds me of the horrible things I did. She is cured of the suppuration but hears very poorly. Also, these radical or semi-radical operations are not as frequent as they used to be. They do not occur in the newer generation. The chronic suppurating ears found in our clinics today are comparatively rare because the acute ear has been cured.

Also, local treatment in chronic suppuration is better, especially since the use of iodine boric powder insufflation combined with capillary suction.

DR. SHERMAN SHAPIRO: I would like to ask if the doctor reported all the cases operated upon by the Bondy method, including those, if any, that had to be reoperated later on with more radical procedure, or only those cases that showed improvement in hearing?

DR. L. J. WALLNER (closing): I want to thank Dr. Beck and the others for their discussion. Regarding the statement that some of the hearing may have been from the other ear: In two it was the better ear that was operated upon; in the other ear the whisper was heard at barely six inches or less. In the case of the complete radical, in the ear operated upon the whisper was heard close to the ear with the other ear stopped with the finger. A person with normal hearing in both ears and with both ears stopped with the fingers does not hear the whisper until close to the ear. So far as aftercare in the two cases without skin graft is concerned, in one case it was six months before it was dry; granulations persisted and finally a bridge formed and became fibrous; this was broken down with forceps. After that it became dry. I reported six cases, one patient did not appear. In that case there was no skin graft and these bands had to be removed several times. Dr. Shapiro's question as to whether we showed all the cases, I have not shown all the cases that were done by this method. I chose some with reference to the status of hearing. I do not know of any that have been reoperated upon.

Abstracts of Current Articles

NOSE

Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses.

Courville, Cyril B., and Rosenvold, Lloyd K. (Los Angeles), Arch. Otolaryng., 27:692 (June), 1938.

1. A survey was made of the infectious intracranial lesions secondary to diseases of the nasal air passages and accessory sinuses in a series of 15,000 cases in which autopsy was performed. A total of 62 cases were studied. In all cases sinusitis was associated with otitis media and therefore it is questionable which lesion might have been responsible for the intracranial disease. In 51 cases the intracranial septic lesion was secondary to an infectious rhinal lesion. In one of these cases syphilitic erosion of the base of the skull was responsible for the creation of an atrium for extension of infection into the intracranial space. In three cases the infectious lesions were consequent to bony erosion by tumor (in four other cases a tumor extending into the skull did not result in a septic lesion).

2. In the first part of the study attention was given to the specific types of rhinogenic lesions which may be encountered within the cranial cavity, the possible sources of infections, their pathogenesis, their morbid anatomy and the associated lesions. The possible lesions consequent to diseases of the nasal air passages and accessory air sinuses are pachymeningitis externa and interna, extradural and subdural abscess, dural fistula, septic thrombosis of the cavernous or the superior longitudinal sinus (rarely of other sinuses), septic meningitis and abscess of the brain of a variety of sources, types and locations.

3. Frontal sinusitis was responsible for the intracranial lesion in fourteen cases, extradural abscess in four cases, subdural abscess in six cases and cerebral abscess in only two cases. Septic thrombosis of the superior longitudinal (sagittal) sinus in this series was secondary to frontal sinusitis (two cases). Septic meningitis, on the other hand, was a primary or direct lesion but was usually associated with and evidently consequent to some other frontogenic infectious lesion.

4. Ethmoiditis (eighteen cases) and sphenoiditis (twenty-two cases) were frequently associated lesions, and their most common intracranial complication was septic meningitis, which was present in sixteen and in twenty of the respective diseases. In a number of

these cases both sinuses were affected. Furthermore, in some instances, the association of sphenoiditis and otitis media observed at autopsy made it difficult to determine which lesion was responsible for meningitis. Extension into the cranial space was usually by way of the lamina cribrosa. Rhinogenic thrombosis of the cavernous sinus, present in a higher proportion of cases in other series, was present in only three cases. In all three cases sphenoiditis was present.

5. In three other cases thrombosis of the cavernous sinus was due to infectious lesions (boils or ulcerations) in the region of the nares.

6. Intracranial complications consequent to maxillary sinusitis are rare, only four cases being found in our series. Direct extension with extradural and subdural lesions and with or without abscess of the temporal lobe is the rule. For reasons not entirely clear, maxillary sinusitis of dental origin is more apt to provoke intracranial suppurative lesions than any other type.

7. In general, an acute infectious lesion is more apt to result in intracranial complications than is a chronic one, a situation which differs from that present in otitis media and in mastoiditis, in which chronic disease is more prone to extend intracranially. Even to a greater extent than with otitis media, such rhinogenic complications are apparently much more common in males than in females (4 to 1).

TOBEY.

Radiotherapy of Furuncles of the Upper Lip. (Traitement radiothérapique des furoncles de la lèvre supérieure.)

Irisarri, J. M. (Bordeaux). Rev. de Lar. Otol. Rhin., 58:1087 (Dec.), 1937.

Radiotherapy is strongly recommended for all facial furuncles, especially those of the upper lip and nose. Aside from slight swelling and increase of pain the first day, these desions dry up and disappear within four days.

FENTON.

LARYNX

Bilateral Recurrent Paralyses. (Les paralysies récurrentielles doubles.)

Flottes, L. (Bordeaux). Rev. de Lar. Otol. Rhin., 58:25 (Jan.), 1938.

This exhaustive study reviews the literature completely, and concludes that in the absence of extensive local difficulties in the neck or chest (aneurysm, neoplasm, etc.), bilateral recurrent paralyses are always of central origin.

FENTON.

Sympathetic Innervation of the Larynx. (L'Innervation sympathique du larynx.)

Terracol, J. (Montpellier).

Professor Terracol reports a detailed anatomical, histological and experimental study of the laryngeal innervation, concluding that the motor nerves contain numerous sympathetic fibers and small ganglia. The cervical sympathetic furnishes vasoconstrictor elements, while the superior laryngeal nerve brings about vasodilatation. Periarterial sympathetic fibers react to produce local ischemia particularly in the arytenoid region, even when stimulation occurs in distant sensory nerves in other parts of the body. Intimate sympathetic connections with the thyroid are described.

FENTON.

The Surgical Treatment of Chronic Cicatrical Stenosis of the Larynx.

Schmiegelow, Professor E. (Copenhagen), J. Laryng. & Otol., 53:1-14 (Jan.), 1938.

This presentation is the Semon Lecture before the University of London in 1937. The author reviews his experiences with stenosis of the larynx over a period of 50 years.

Based upon this experience, he believes that the most important feature in the management of the condition is a clear understanding of the cause and the nature of the stenosis. One of his cases, for example, was the result of a foreign body. Even in the presence of complete occlusion by the formation of fibrous tissue, the author believes that every case may be treated by the method which he has previously described and which he here presents again in detail. The method is regarded as being without great danger and the 18 patients included in this study have been cured in from one to ten weeks, the average being about six weeks.

Subglottic Cancer of the Larynx.

Thomson, Sir St. Clair (London), J. Laryng. & Otol., 52:803-816 (Dec.), 1937.

This study demonstrates that subglottic cancer is much less frequent than either the intrinsic or the extrinsic forms. Early diagnosis is more difficult. The choice of surgical treatment is not so definite and does not give such good results as in intrinsic cancer proper, though they are better than in extrinsic disease.

The lesion is generally well advanced before it reaches the cords, causing change of voice. The early symptoms may be intermittent disturbances of voice or partial paresis of one cord. It should be suspected whenever there is any restriction in movement of one cord, especially when this occurs without a visible lesion on its surface.

When the anterior commissure has been invaded, a complete laryngectomy is indicated. The extent of a subglottic growth cannot be correctly judged until the larynx is split, when the extension is generally found to be greater than was anticipated. For patients whose age, health or prejudice forbids a complete laryngectomy and for those in whom it does not promise success, treatment by radiation should be given. In this connection, it should be remembered that the full extent of these growths is often misjudged and that some failures may be attributed to this cause.

Double Foreign Body in the Larynx. (Corps étranger double dans le larynx.)

Sercer, A., and Gisic, B. (Zagreb). Bronchoscopie, Oesophagoscopie et Gastroscopie, 2:110 (April), 1938.

In general, multiple foreign bodies in the air passages consist either of fragments of the same body, or similar bodies, such as nails or tacks aspirated during the same accident. In this case, a 13-months-old child, while playing on the floor, began suddenly to choke and to cry. When seen five hours after the accident, dyspnea was extreme and tracheotomy had to be performed with resulting complete respiratory relief. Lower tracheo-bronchoscopy was negative but on laryngoscopy a large grape seed was found and was expelled in toto. However, decanulation could not be successfully accomplished, and two weeks later, upper tracheoscopy under anesthesia revealed a hard, black subglottic foreign body surrounded by granulations, which on removal, proved to be a piece of coal about 7 mm. long. Two days later the child was successfully decanulated.

KLAWANS.

ESOPHAGUS, TRACHEA AND BRONCHUS

The Pathology and Treatment of Carcinoma of the Bronchus.

Ormerod, F. C. (London), J. Laryng. & Otol., 52:733-745 (Nov.), 1937.

This article is based on 100 consecutive cases of bronchial carcinoma in each of which a portion of the growth was removed through the bronchoscope and its malignant nature demonstrated histologically. The author analyzes the locations of these tumors and discusses the differential diagnosis from various other pulmonary lesions.

Various methods of treatment are discussed and operative results analyzed.

The article is illustrated with numerous photomicrographs and radiographs.

Integration of Clinical and Roentgenologic Findings in the Diagnosis of Carcinoma of the Esophagus.

Farrel, John T., Jr., Radiology, 30:4 (April), 1938.

One hundred cases were studied. Cancer occurs three times more frequently in the lower and middle than upper third of the esophagus, and is twelve times more frequent in males than females. Eighty-six per cent of the patients were over fifty years of age. Familial malignancy plays no role. The influence of syphilis is of no apparent import.

An attempt was made to correlate clinical and radiologic findings to reveal possible signs which might lead to more early diagnoses.

Symptoms were present less than six months in 58 per cent, less than a year in 87 per cent of the cases. The majority of the lesions were advanced at the time of observation.

Dysphagia was the most frequent initial symptom. Pain is a later accompaniment. It does not accompany phrenospasm, diverticulosis, or varices of the esophagus.

Malignancy was suspected in 95 per cent of the cases at the time of first roentgen study.

Surgery offers the best method for coping with the condition, and this must be done early.

JORSTAD.

Esophagofissure (Low External Esophagotomy). (L'oesophagofissure. L'oesophagotomie externe basse.)

Dr. Belinoff (Sofia). Bronch. Oesoph. et Gastr., p. 73, (April), 1938.

The author reports two cases in which his external esophagotomy was done. In the first case, a six-year-old, gastrostomized child, with an impassable esophageal stricture from a lye burn, he exposed the stricture but was unable to penetrate it. Healing was uneventful. In the second case, he extracted through an external operation a denture which had been impacted in the esophagus for two weeks and which had resisted endoscopic removal. The patient recovered and the wound healed in five weeks.

He advises low cervical esophagotomy as an approach to the thoracic esophagus, inserting a long bladed Killian nasal speculum through a long (5 cm.) incision through the esophageal wall to expose the lower regions down to the level of the tracheal bifurcation. The esophageal wall is sutured to the skin and the wound is allowed to close by granulations.

KLAWANS.

Emergency Pneumothorax in Atelectasis Caused by an Intrabronchial Foreign Body. (Le pneumothorax. L'Indication d'urgence dans les atélectasies causées par un corps étranger intra-bronchique.)

Capurro, Dr. F. G. (Montevideo, Uruguay). Bronchoscopie, Oesophagoscopie, et Gastroscopie, 2:94 (April), 1938.

A 21-months-old child, while eating melon, suddenly developed alarming choking. Thirteen hours later the child was hospitalized with marked cyanosis, toxemia, stupor, and fever. A clinical diagnosis of subglotic obstruction with atelectasis of the right lung, dextro-cardia, and emphysema of the left lung was made. Laryngoscopy was negative, and a bronchoscopist was not available at the time. X-ray examination confirmed the chest findings and disclosed a markedly displaced mediastinum.

Because of the alarming state of the child, it was suggested that a pneumothorax on the right side might relieve the mediastinal displacement, restore more normal circulation and thus relieve cyanosis. This was done and cyanosis and stupor immediately disappeared, the pulse rate dropped and the patient improved markedly. After eight hours, the alarming symptoms reappeared and were again relieved by pneumothorax only to reappear in six hours. The child died before bronchoscopy could be done and at autopsy, a vegetal foreign body, a seed, was found in the right main bronchus producing complete obstruction.

The author suggests that in cases of imminent asphyxia due to atelectasis resulting from bronchial obstruction by a foreign body, prolonged palliation may be obtained by means of a pneumothorax, thus allowing time for extraction of the foreign body.

KLAWANS.

EAR

Facial Palsy of Otitic Origin.

Persky, Abram H. (Philadelphia), Arch Otolaryng., 27:395 (April), 1938.

Conclusions to be drawn from this study:

1. While facial paralysis may occur preoperatively in cases of either acute or chronic suppurative otitis media, it is of greater significance and graver portent, both as to cure and as to the possibility of intracranial complication, in the latter group, especially in the presence of cholesteatoma. Hence, while one may be tempted to temporize in cases in which the involvement is acute, the situation is urgent in cases of chronic involvement.

2. In cases in which the condition is simple and acute, thorough simple mastoidectomy should be the operation of choice. In our

series the results obtained from complete radical mastoidectomy with exposure of the facial nerve and subsequent splitting of the sheath did not compare favorably with the results of simple mastoidectomy. Moreover, the latter procedure should be reserved for cases in which facial palsy has occurred postoperatively.

3. Any facial palsy following operative procedure calls for an immediate exploratory operation, with an attempt to localize the site of injury and expose the injured portion of the nerve. Treatment should then be by simple drainage in cases in which there is a serous or a hemorrhagic exudate, by removal of a spicule of bone, by splitting of the sheath, by immediate repair of the severed neurons by approximation or by autoplastic graft from either proximal or distal nerves, according to the type of lesion found.

4. Finally, the duration of the palsy may be an index of the prognosis. In cases in which the palsy is of short duration, favorable results may be expected of the indicated treatment. However, when the palsy has been present for a considerable period and reactions of degeneration have set in, the prognosis is necessarily more guarded, the results depending on the success of repair or other surgical procedure on the nerve itself.

TOBEY.

Impaired Hearing for High Tones

Crowe, S. J., and Guild, S. R. (Baltimore). Acta Oto.-Lar. 36:2:138 (Jan.-Mar.), 1938.

The authors point out that middle ear lesions resulting from occlusion of the pharyngeal orifice of the eustachian tube usually cause greater impairment for high than for low tones. They found in 20 cases, after operative removal of adenoid hyperplasia, that limited and carefully screened use of radium emanation at the mouth of the tube produced excellent results, but the permanence of hearing improvement cannot as yet be predicted.

FENTON.

Electrophysiology of Hearing

Hallpike, C. S. (London). Acta Oto.-Lar. 36:2:145.

Summarizing a series of experiments, Hallpike has decided that the cochlear response occurs through a vibrating, resonant structure, probably Reissner's membrane, and not from the hair cells of the organ of Corti. Normal response may occur when the hair cells have degenerated, but is blocked when the hair cells are normal but the perilymph circulation has been obstructed.

FENTON.

Radiography of the Eustachian Tube. (Étude radiographique de la trompe d'eustache.)

Heimendinger, E. (Strasbourg). Rev. de Lar. Otol. Rhin., 58:303 (Mar.), 1938.

Silk eustachian bougies containing bismuth or lead can be inserted in the tube by the usual technique, permitting accurate visualization of the course of the tube with the film in the vertex-chin position of Hirtz. Obstructions at the isthmus are readily observed by this technique.

FENTON.

The Air Cells of the Petrous Portion of the Temporal Bone.

Wilson, J. Gordon; Gaardsmoe, John P., and Anson, Barry J. (Chicago), J. Laryng. & Otol., 52:746-751 (Nov.), 1937.

This anatomical study of the temporal bone reveals several points important to the surgeon. Cells of the pyramid have their origin from two sources: the hypotympanic space and a prolongation of the mastoid cells. The cells of the former group are much less accessible than the others, owing to their relationship to the carotid canal. The latter are relatively easily uncovered by mastoid opening.

It is extremely necessary for the surgeon to have knowledge of the location of the infection, but locating the lesion is not always easy. Roentgenograms are not particularly helpful. The general history of the case and the location of the pain are more to the point. The pain typical of petrositis is homolateral; it is orbital and temporal, that is, confined to the distribution of the ophthalmic division of the trigeminal nerve.

Endaural, Antauricular Surgical Approach to the Temporal Bone.

Lempert, Julius (New York), Arch. Otolaryng., 27:555 (May), 1938.

Endaural, antauricular operation on the temporal bone is not performed through the external auditory canal but through a widely opened mobile membranous and extracartilaginous endaural window which, together with the auricle, may be moved and displaced in every desirable direction over the temporal bone.

A new anatomic landmark is described and designated as the antauricular, suprameatal membranous triangle. The surgical employment of this triangular membranous space makes possible the endaural, antauricular approach in all surgical attacks on the temporal bone.

Many technical difficulties encountered with the postauricular surgical approach to the temporal bone, some of which are often insurmountable to the less experienced and conquerable only by the

extremely skillful surgeon, are naturally solved by the very technique of the newly described endaural, antauricular surgical approach.

A careful comparative study of the postoperative course, the convalescence, the period of healing and the ultimate end results obtained in the 1,780 consecutive cases in which operation was performed by me through the endaural, antauricular surgical approach, together with the careful observation of the many advantages enjoyed by my patients as a result of the adoption of this new technique, more than justifies my abandonment of the postauricular approach to the temporal bone.

TOBEY.

On Bone Formation in the Scala Tympani of Otosclerotics.

Nager, F. R. (Zürich), and the late J. S. Fraser (Edinburgh), J. Laryng. & Otol., 53:173-180 (Mar.), 1938.

The author describes six observations of new bone formation in the scala tympani of subjects with otosclerosis. The otosclerotic process in all these observations was greatly extended and penetrated even into the newly formed lamellar bone. The change in the scala tympani is considered a consequence of the irritation by the otosclerotic process of the labyrinthine wall and has not been found in any other bone disease.

On the Origin of the Wever-Bray Response and on an Electrotherapy of the Ear.

Kupfer, E. (London), J. Laryng. & Otol., 53:16-31 (Jan.), 1938.

This article reviews a series of previous reports upon an electrochemical theory of hearing. The author feels that the electroacoustic effect and the Wever-Bray phenomenon may be explained by the disturbance and displacement of Freundlich's electrical double layers at phase boundaries of liquids and solids in the cochlea. These double layers of potential may be displaced by movements of the stapes as well as by an external E. M. F. Tinnitus interpreted as an electrical phenomenon was said to have been neutralized in many cases by electro-therapy.

Osteomyelitis of the Petrous Pyramid.

Kelemen, G. (Budapest), J. Laryng. & Otol., 53:113-126 (Feb.), 1938.

The author describes two cases of osteomyelitis of the temporal bone and discusses the findings at autopsy. His findings appear to support the contention of Otto Mayer that the infection never emanates from the marrow spaces, even when the tip is not pneumatized; it begins invariably with the pneumatized cells. He points out that involvement of the non-pneumatized petrous tip must be an osteomyelitis, displaying all the stormy and serious symptoms of this disease.

Books Received

Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla.

Dr. Nils Ringertz, from the Department of Pathology, Sabbatsberg's Hospital, Stockholm (Chief: Professor H. Bergstrand). Paper. Royal 8vo. of 405 pages with 109 illustrations. Helsingfors. Mercators Tryckeri. 1938.

This monograph in English is a complete text on the subject. The material has been gathered from the clinics of Stockholm and contains the cases of Holmgren, Berven and Öhngren, some of which have been the subject of previous detailed communications by these authors. It comprises in all 391 cases, which are abstracted at the back of the book.

Following reviews of the anatomy, histology and embryology and of the literature, the individual types of tumor are taken up seriatim. A comparison is made in each case between the author's findings based upon his own material and the opinions of others gathered from the literature. Each paragraph is illustrated with exceedingly good photomicrographs.

Studies on the Physiology of the Middle Ear.

J. Grandson Byrne, M.A., M.D., LL.D. Cloth. Demy 8vo. of 289 pages with 54 illustrations in the text. London: H. K. Lewis & Co., Ltd., 1938. Price 18s. net.

This volume is a report of a long series of laboratory experiments conducted by the author over a period of several years. It is in two parts.

The first deals with reactions of the membrana tympani following experimental section of various pathways. Two chapters are devoted to the tonus of the Vth and VIIth cranial nerves and another to the effect upon the auditory tube of lesions of the efferent paths which supply the dilatator tubæ auditivæ. There is also a study of the microscopic features of the muscles of the middle ear in the dog and cat and their physiological significance.

The second part describes a series of stimulation experiments upon various nerves and pathways concerned with the middle ear.

This should be an invaluable reference book to the otologist interested in the clinical physiology of the ear.

Die Missbildungen und Anomalien der Nase und des Nasenrachenraumes. (Deformities and Anomalies of the Nose and of the Nasopharynx.)

Dr. Walther Stupka, Privatdozent for Otolaryngology in the University of Innsbruck and Head of the Department of Oto-rhino-laryngology in the General Hospital of Vienna-Neustadt. Paper. Royal 8vo. of 318 pages, with 153 illustrations. Vienna. Julius Springer, 1938. Price 36 RM, bound 38.70 RM.

This is a monograph written in German. The first two chapters are given over to a description of the embryology of the nose and the points at which deviations from the normal may occur. There follows a comprehensive chapter on experimental teratology, chiefly in amphibians.

The material relative to malformations and anomalies of the nose and of the naso-pharynx is presented in great detail. The illustrations are excellent.

Notice

Casselberry Prize Fund

The sum of $500.00 having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, said sum is now available in part or as a whole, for a prize award, decoration, or the expense for original investigation or research in the domains mentioned above. Theses or reports of work done must be in the hands of the Secretary, Dr. James A. Babbitt, 1912 Spruce street, Philadelphia, Pa., before February 1st of any given year. Any further information desired will be gladly furnished by the Secretary.